ANNUAL MEETING: NEWCASTLE UPON TYNE, JULY 10-19

DOES NOT CIRCULATE

BRITISH MEDICAL JOURNAL

JOURNAL OF THE BRITISH MEDICAL ASSOCIATION

SATURDAY FEBRUARY 16 1957

UNIVERSITY OF MICHIGAN
MAR 7 1957
MEDICAL LIBRARY

TABLE OF CONTENTS

Medical Aspects of Boxing, Particularly from a Neurological Standpoint
MACDONALD CRITCHLEY, M.D., F.R.C.P., F.A.C.P. 357

Further Observations on the Medical Aspects of Amateur Boxing
J. L. BLONSTEIN, M.R.C.S., L.R.C.P., and EDWIN CLARKE, M.D., M.R.C.P. 362

A Fatal Injury at Boxing (Traumatic Decerebrate Rigidity) MILROY PAUL, M.S., F.R.C.S. 364

Antigenic Activity of British Poliomyelitis Vaccine REPORT TO THE MEDICAL RESEARCH COUNCIL 366

Poliomyelitis Studies in an Isolated Scottish Community A. D. MACRAE, M.D., DIP.BACT. 368

Deaths from Poliomyelitis Among British Doctors
RICHARD DOLL, M.D., M.R.C.P., and A. BRADFORD HILL, C.B.E., F.R.S. 372

Investigation of Non-ulcer Dyspepsia by Gastric Biopsy A. WYNN WILLIAMS, M.D., D.C.P.(Lond.), FELICITY EDWARDS, B.M., M.R.C.P., T. H. C. LEWIS, B.M., and N. F. COGHILL, M.A., M.B., M.R.C.P. 372

Respiratory Virus Infections in Hospital and in General Practice, 1954–6
N. R. GRIST, M.B., B.SC., M.R.C.P.Ed., R. G. SOMMERVILLE, M.B., and H. G. CARSON, F.I.M.L.T. 378

Infection with Sendai Virus in an Outbreak of Respiratory Illness G. B. BRUCE WHITE, M.R.C.S., DIP.BACT., P. S. GARDNER, M.B., DIP.BACT., and R. E. HOPE SIMPSON, M.R.C.S. 381

Suxamethonium Chloride Administration and Post-operative Muscle Pain
D. D. B. MORRIS, M.B., F.F.A. R.C.S., D.A., and C. H. DUNN, F.F.A. R.C.S., M.R.C.S., D.A. 383

Generalized Arterial Calcification of Infancy A. C. HUNT, M.D., and D. G. LEYS, D.M., F.R.C.P. 385

MEDICAL MEMORANDA

Primary Dislocation of Left Shoulder-joint in Boxing. R. S. WOODS, M.D., F.R.C.S. 387
Agranulocytosis During Treatment with an Amoebicide. H. F. HAHNDEL, M.D., and L. PREGER, M.B., D.Obst.R.C.O.G. ..387

GENERAL ARTICLES AND NEWS

Fatal Hospital Falls. GAVIN THURSTON, M.R.C.P., D.C.H. 396
Medical Ethics in Wartime 397
BOOK REVIEWS 388
MEDICAL NOTES IN PARLIAMENT 410
VITAL STATISTICS 411
MEDICAL NEWS 413

OBITUARY

D. R. MacCalman, M.D., F.R.C.P.Ed.; W. H. Wilson, D.M.; H. M. Shelley, F.R.F.P.S.; R. W. D. Leslie, C.B., L.R.C.P.I.; Rufus C. Thomas, F.R.C.S.Ed., F.R.C.O.G.; W. N. Gray, M.B.; G. W. Lloyd, M.D.; G. O. Montgomery, M.B.; C. L. Gimblett, M.D., F.R.C.S. 405-9

LEADING ARTICLES

Viruses Made to Order 390
Non-ulcer Dyspepsia 391
Boxing Injuries 392

ANNOTATIONS

Bilirubin 393
Doctors' Diseases 394
Health Service Supplementaries 394
Compound Aspirin and Steroid Tablets 395
Hungarian Medical Refugees 395

CORRESPONDENCE

Hungarian Medical Refugees. S. WAND, M.B. 398

SUPPLEMENT

Full Contents on First Page of Supplement
Joint Consultants Committee 65
Central Consultants and Specialists Committee 66
Correspondence:
Unity of Purpose. C. A. SHARPLES, M.B.; G. J. SOPHIAN, F.R.C.S. 69

CORRESPONDENCE—*contd.*

Prednisone and Prednisolone. F. DUDLEY HART, M.D. 398
Prednisone and Nasal Allergic Disease. H. J. SHAW, F.R.C.S. 398
Safety Factor in Car Design. WILLIAM GISSANE, F.R.C.S. 399
Tuberculosis in Glasgow. G. L. COX, M.D. 399
Incidence of Venereal Disease. G. H. KNIGHT, M.D., and W. FOWLER, M.D. ..399
The Hurt Brain. E. E. PREBBLE, M.D. ..399
Asphyxia Due to Faulty Apparatus. F. J. R. STONEHAM, F.F.A. R.C.S. 400
Acute Appendicitis. E. C. ATKINSON, M.B.; J. R. HUGHES, F.R.C.S.Ed.; R. C. MAY, M.R.C.S. 400
Abnormal Haemoglobin. J. A. M. AGER, M.B. 401
The Black Sheep. J. H. HUDSON, M.B.; RAYMOND GREENE, D.M. 401
Fatal Reactions to Local Anaesthetics. A. C. PENMAN, M.D. 401
Mild Endogenous Depression. R. THOMPSON, M.B., D.P.M. 401
Letters on other subjects on pp. 402–5

BRITISH MEDICAL ASSOCIATION, TAVISTOCK SQUARE, LONDON, W.C.1

No. 5015, WEEKLY, PRICE 3/-

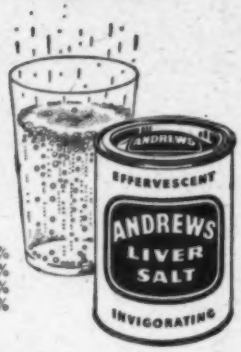
Copyright

REGISTERED AS A NEWSPAPER

When a gentle laxative is needed . . . Andrews Liver Salt may be indicated

Effervescent

Pleasant-tasting



APPROX. COMPOSITION

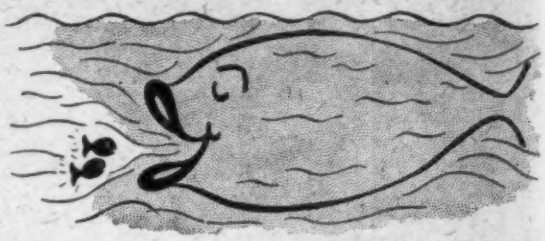
Tartaric Acid	23%
Sodium Bicarbonate	23%
Magnesium Sulphate (B.P.C.Exice) ...	17%
Sugar	37%

An 8oz. tin for clinical trial will gladly be sent on request

SCOTT & TURNER LTD.

ANDREWS HOUSE · NEWCASTLE UPON TYNE

The original easy-to-take, two-in-one tablet



for the prompt and prolonged relief of **ASTHMA**

ISO-BRONCHISAN was first produced in 1953 and offers a fresh approach to the problem of effective asthma control. The tablets, which are pleasant to take, have a coating containing easily dissolved Isopropyl-Nor-Adrenaline, a most potent bronchodilator, which, when absorbed by the sublingual route, produces the prompt relief of bronchospasm. The rest of the tablet, when swallowed, releases Ephedrine and Theophylline in balanced proportions and these, slowly absorbed along the alimentary tract, ensure long sustained antispasmodic action on the bronchial smooth muscle.

ISO-BRONCHISAN

Prescribable on Form E.C.10.

SILTEN

Each tablet contains Isopropyl-Nor-Adrenaline (Isoprenaline) sulphate gr. ½; Ephedrine hydrochlor. gr. 2/5; Theophylline gr. 2.
In tubes of 20 tablets and bottles of 100 tablets.
Tablets containing smaller quantities of the effective ingredients are now available for use in Paediatrics.

Samples and literature available on request.

SILTEN LIMITED · SILTEN HOUSE · HATFIELD · HERTS · ENGLAND

RHEUMATISM
AND KINDRED AILMENTS

Harrogate, the largest treatment centre in Great Britain, is actively engaged in providing all types of physical treatment in connection with the rheumatic diseases and all types of physical rehabilitation. Extensive alterations have taken place, including the equipment of the establishment with DEEP POOL THERAPY, medical gymnastic facilities and occupational therapy.

HARROGATE

Treats both private patients under its All-inclusive Treatment Scheme, and National Health patients.

Medical enquiries as to cost, and how treatment can be obtained, will be welcomed by :

E. ROBERTS, Manager
Section I

THE ROYAL BATHS
HARROGATE

POST GRADUATE STUDY

☞ **Are you preparing for any higher Medical or Surgical Examination?**

Send below for free valuable publication

"GUIDE to MEDICAL EXAMINATIONS"

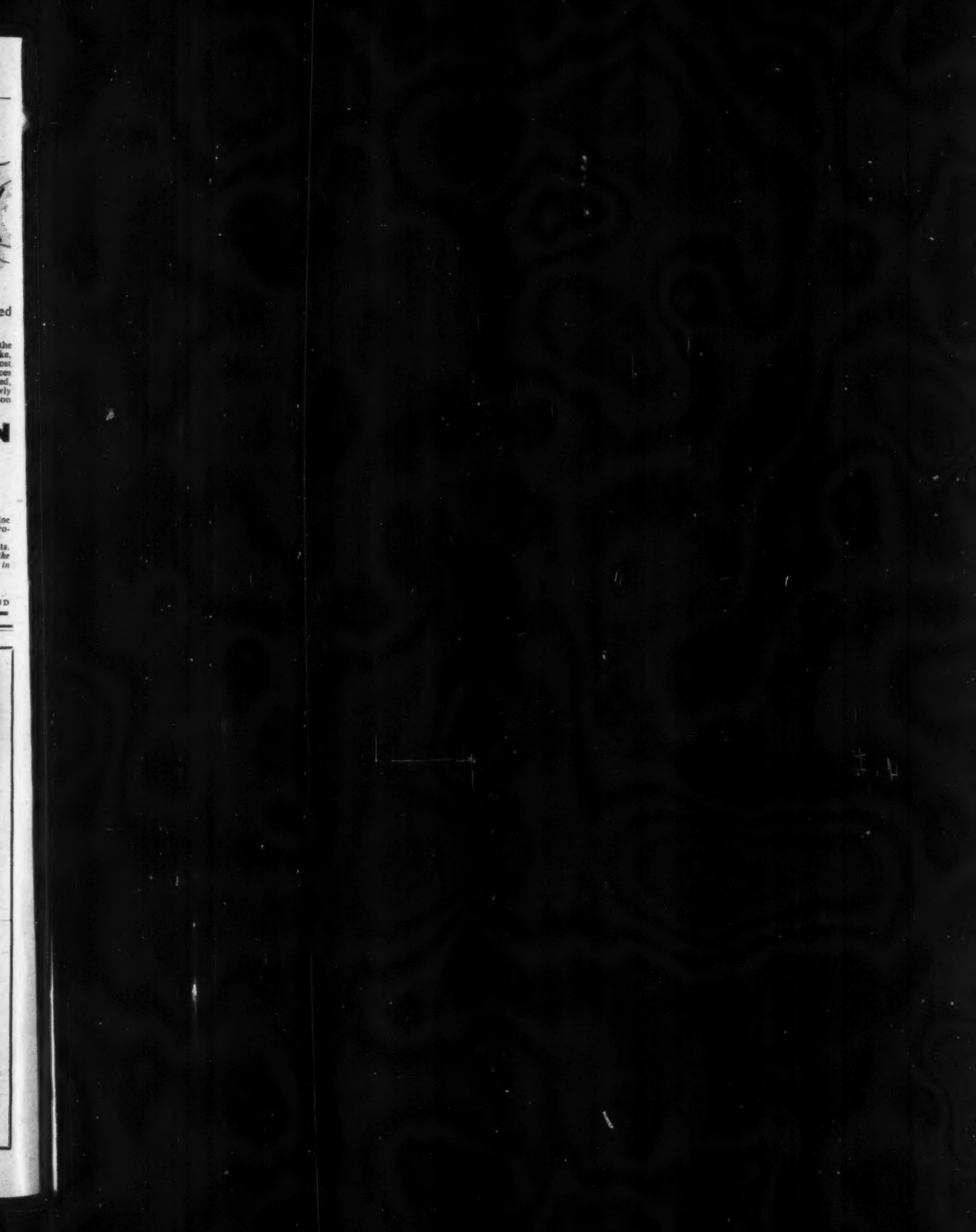
PRINCIPAL CONTENTS

The F.R.C.S. England, Edinburgh and Ireland.
The M.R.C.P. London, Edinburgh and Ireland.
The M.D. Thesis of all Universities.
The F.F.A. and Diploma in Anaesthetics.
The Diploma in Tropical Medicine.
Diploma in Ophthalmology.
Diploma in Psychological Medicine.
Diploma in Child Health.
Diploma in Physical Medicine.
Diploma in Public Health.
Diploma in Pathology.
Diploma in Radiology.

You can prepare for any of these qualifications by postal study. We specialize in Post-graduate tuition.

Write stating examination in which interested to

The Secretary,
MEDICAL CORRESPONDENCE COLLEGE,
19 Welbeck Street, London, W.1



BRITISH MEDICAL JOURNAL

LONDON SATURDAY FEBRUARY 16 1957

MEDICAL ASPECTS OF BOXING, PARTICULARLY FROM A NEUROLOGICAL STANDPOINT*

BY

MACDONALD CRITCHLEY, M.D., F.R.C.P., F.A.C.P.

Neurologist, King's College Hospital; Physician, National Hospital, Queen Square, London

Traumata arising in the course of boxing contests or during a pugilistic career are diverse in character. Whether they are to be regarded as commendably rare or as alarmingly common is a matter of opinion. It is sometimes claimed that boxing *per se* causes no more injuries or fatalities than some other sporting events. Thus Johansen (1955), of Oslo, has analysed a series of 6,057 sport injuries occurring during the period 1946–8: of these, boxing, which came tenth out of a list of 14 athletic pursuits, was held responsible for only 100 accidents (1.65%). This contrasts with the numbers attributed to skiing (1,784, or 29.4%) and to football (1,320, or 21.8%).

One important distinction, however, distinguishes boxing from most other forms of athleticism. Injuries are coincidental in other sports, but in boxing the aim and object—explicit or implied—is to render the opponent *hors de combat*. Traumata are therefore not so much regretted as regrettable.

This paper makes no plea either for or against pugilism. Amateur boxing can certainly fulfil many useful purposes, sociological as well as personal. Whether the benefits of amateur boxing outweigh the drawbacks is a question for others to decide; while some may well consider that professional pugilism forms a problem all of its own. The purpose of this communication is merely to enumerate some of the principal ill effects which have been ascribed to boxing, with particular reference to the neuro-psychiatric deterioration which may develop late in the career of a prize-fighter.

The "Knock-out"

The "knock-out"—so dramatic a spectacle in professional contests—entails the deliberate and violent production of a state of motor hypotonus and helplessness coupled with a severe—if short-lived—disturbance of consciousness. As a rule this knock-out phenomenon is the result of a violent blow to the point or side of the chin. The victim collapses heavily and remains immobile or inert for a matter of seconds or minutes. The unconsciousness produced by the knock-out blow is clinically rather unusual, being as a rule very abrupt in onset, quite short in duration, and comparatively complete in recovery. In falling, the head may forcibly strike the ground, thereby producing a second and direct injury to the head which of itself may complicate the clinical picture. If the boxer is rendered so helpless as to be unable to regain the erect stance within 10 seconds, he is "counted out." After a knock-out blow most boxers are able to leave the ring within a minute or two, though ordinarily with the help of their seconds. Longer periods of powerlessness, or of unconsciousness, are by no means unknown, however.

According to the sporting fraternity there are causes of a knock-out other than a blow to the chin. Thus there is the "solar plexus knock-out," which is not ordinarily attended by unconsciousness. Jokl (1941) believed that this type of injury is identical with the experimental *Klopfversuch* studied so thoroughly by Goltz (1863). There is also a "heart knock-out" (or commotio cordis), which on occasion has proved fatal. Blows to the side of the neck may provoke what physicians may regard as a "carotid sinus knock-out." The physiological mechanism of this lesion has been particularly associated with the work of Hering (1927). According to Boigey (1938), the right side of the neck is more sensitive to blows than the left, so far as the carotid sinus is concerned. There are, in addition, certain types of foul punch which are apt to produce a knock-out. These include a downward chop to the nape of the neck (rabbit punch); blows "below the belt" (a euphemistic term for direct injury to the testicles, which is apt to produce much pain, shock, and occasionally unconsciousness); and "kidney punches," which have been known to provoke haematuria and even renal lacerations.

The ordinary classical knock-out blow is, however, an indirect head injury, where the point of impact of the fist is to the "button"—that is, the point of the chin. The violence is transmitted up and back to the base of the skull. The exact mechanism of the loss of consciousness and tonus has been a source of controversy. Sherrington's (1906) description deserves quotation: "The 'knock-out blow,' where the lower jaw conveys concussion to the otocyst, reduces in a moment a vigorous athlete to an unstrung bulk of flesh whose weight alone determines its attitude, if indeed a reactionless mass can be described as possessing attitude at all." It is interesting to note that Sherrington did not mention disordered consciousness. In other words, concussion of the otocyst is—in his opinion—the essential factor of the knock-out, leading to an abrupt hypotonus of vestibular origin. Somen (1913–14) also implicated the inner ear; and, according to Somen, Babinski concurred. La Cava (1952) believed that a violent blow to the mandible involves nerve structures immediately behind the angle of the jaw, and leads to an abrupt drop in blood pressure. Denny-Brown and Russell (1941) visualized a sudden stimulation of vasomotor centres leading to a temporary depression in cardiac, vasomotor, and

*Being the opening paper at a discussion upon this topic held at the Chelsea Clinical Society on November 13, 1956.

respiratory function. Jokl (1941), like Winterstein (1938), also imagined a condition of mechanical medullary dysfunction which may be complicated by concussion due to the direct effect of the fall.

More precisely Jokl and Winterstein (and also Gonzales, Vance, and Helpen, 1937) believed that a blow to the chin produces a two-phasic jerk which involves the medullary centres. In the first place, the clivus and the anterior edge of the foramen magnum impinge violently against the pons and upper medulla; while during the second phase, by virtue of a *contre-coup*, the medulla bounces back against the occipital bone and the posterior edge of the foramen magnum. Gonzales *et al.* described this double impact as "oscillation" of the brain.

The investigations made by Larsson *et al.* (1954), though not conclusive, are rather against any theory that a drop in blood pressure is the *modus operandi* of the knock-out, suggesting that the factors responsible are essentially concussive in type.

Other hypotheses about the cause of the knock-out include that of Flint (1920), who imagined that a distortion of the jaw mechanically impinges upon the carotid artery and so produces a temporary cerebral anaemia. Pfeiffer (1922) implicated a mechanical and short-lived obstruction of the jugular vein.

Amnesias due to Boxing

Just as different types of knock-out can be isolated, so too one can identify a number of different types of amnesia or loss of memory in association with boxing. Thus there may be a period of retrograde amnesia immediately preceding the spell of unconsciousness due to a knock-out. Sometimes there is a more lengthy period of retrograde amnesia, so that the boxer afterwards has no recollection whatsoever of any events of that particular contest. Indeed, he may not be able to remember that the fight even began. This amnesia is of course permanent. Then there are the anterograde amnesias where the pugilist goes on fighting after a knock-out, but without subsequent recollection thereof. This is the groggy-state automatism. Quite independent, of course, is the gross day-to-day memory-defect of the dementing punch-drunk victim. These phenomena are described below.

Other Injuries from Boxing

It does not come within my province to describe in detail the various surgical injuries which have been reported as consequent upon amateur or professional boxing. Some of them are admittedly quite rare, such as ruptured muscles, fractured arm, arteriovenous aneurysm, direct damage to a single nerve—for example, the facial nerve or the long thoracic nerve of Bell (the "commotio nervi" of Jokl). Quite unspecific is the occurrence of bouts of vomiting ("athlete's sickness") which may follow any violent physical exertion. Other injuries are so commonplace as to constitute the ordinary occupational marks of an experienced pugilist. Here belong such physical signs as the cauliflower ear, the flattened nose, hypertrophied superciliary ridges, and arthritis of the first metacarpo-phalangeal joint.

Injuries to the eyes are much more serious and more variegated in character. They have been well described by Doggart (1951, 1955) in this country and by Favory and Sédan (1951) in France. Just how common they are in the course of a boxing career has not been made clear. Among the injuries listed by Doggart are, first, damage to the *outer eye*. Here belong trauma to the conjunctiva and to the cornea, folding or rupture of Descemet's membrane, and scleral rupture. Secondly, a great diversity of lesions of the *inner eye* may occur. These include tears of the iris, perhaps involving the sphincter muscle of the pupil. Persistent hypotony may result from lesions of the ciliary body. Chronic iridocyclitis may develop, and go on to a phthisis bulbi. Accommodation may be paralysed. The lens of the eye may be subluxated or dislocated. Cataract is not uncommon. Haemorrhage into the vitreous may take place, and, though it often resolves satisfactorily, it may lead to a retinitis proliferans. Retinal detachment, which may be bilateral, is a disastrous complication, for it is only rarely amenable to surgery. Cystic and pigmentary changes at the macula and retinal or choroidal haemorrhages may also occur.

Doggart draws special attention to irregular patchy choroidal degeneration in boxers, a feature which he speaks of as "cauliflower choroids." Optic atrophy, avulsion of the optic nerve, haemorrhage into the nerve sheath, and recurrent congestive glaucoma are among some of the other disastrous sequelae that have been described as due to boxing injuries. Doggart finally enumerates injury to the neighbouring structures: fractures of the orbit, periosteal thickening, orbital emphysema, damage to the lacrimal gland or duct. Implication of any of the ocular muscles may occur, especially the superior and inferior obliques.

Boxing Fatalities

Injuries sustained in the ring have ended fatally. Just how the recorded figures strike one depends mainly upon the prejudices and emotions of the reader. Thus, Blonstein and Clarke (1954) have spoken of six deaths over the course of seven years' experience—all in the course of amateur boxing. Kaplan and Browder (1954) have had 26 years' medical experience of the professional ring and have assisted at the necropsy study of five cases. Jokl (1941) went into the published statistics in some detail. He said that Flint (1920) had stated that 124 fatal cases had been recorded up to that time. Since then Jokl had been able to collect another 41 cases between 1920 and 1938. According to Steinhaus (1950b), in the four years prior to 1950 42 professional boxers and 22 amateurs had died from occupational injuries. While the total to the end of 1956 is uncertain, we can say that, up to 1950, at least 207 fatalities had been recorded.

Most of the deaths were due to subdural haemorrhage; some to intracerebral bleeding. At times a heavy fall on the back of the head in the ring was held responsible, rather than blows to the face by a gloved fist. In some cases necropsy demonstrated an innate and unsuspected thinness of the cranial bones. The youthful age of some of the victims is striking—for example, 16 years (case of John Shoddy, 1951); 15 years (Weimann, 1931; Berg, 1933; Kappi, 1938), and 13 years (case of David Zimmerman, 1951). Quite a number of these fatalities had occurred in amateurs and not professionals. In some of them, death had occurred from a blow struck in the first round of a contest.

Besides these deaths from haemorrhage, there are the rarer cases of "reflex death" and "fatal commotio" which may or may not be associated with status thymolymphaticus.

The Punch-drunk State

To a neurologist the chief interest centres in those experienced boxers, whether amateurs or professionals, who have undergone gradual physical and mental deterioration as a direct accumulated result of their careers. This is the condition popularly termed punch-drunkenness. We do not know who it was first coined the term "punch-drunk." Probably it was a layman. Its introduction into the medical literature dates from the paper by Martland in 1928. Since that time it has established itself in medical terminology, and a not inconsiderable literature has developed. The term has also become well known to the laity, and in sporting circles certain pejorative—almost derisory—connotations have become attached both to this particular word and to its synonyms "punchie," "goofy," "slug-nutty," "paper dolls," "stumblebum," and "slap-happy." For such reasons attempts have been made to introduce a more esoteric and less offensive expression. Thus we have "traumatic encephalopathy" (Parker); "traumatic encephalitis" (Ravina); "dementia pugilistica" (Millspaugh); "chronic brain injury after boxing" (Bøje). If we really require a suitable scientific alternative to "punch-drunkenness" we may perhaps adopt the

term "chronic progressive traumatic encephalopathy of boxers."

Up to date, my own neurological experience has comprised a series of 69 cases of chronic neurological disease in boxers. Many of these—perhaps the great majority—should be looked upon as examples of punch-drunkenness, either early or well established. In only a comparatively few cases does legitimate doubt occur about the possibility of there being some coincidental and non-related nervous or mental disease—for example, epilepsy; psychopathy; presenile dementia; cerebral arteriopathy.

The details of the boxing careers in punch-drunk patients are significant. Punch-drunkenness is much commoner among professionals than among amateurs. It is commoner in second-rate or third-rate performers than in the intelligent scientific exponent with a championship title. It is very rare in coloured boxers. As fighters, the punch-drunk ex-pugilists have been slow on their feet rather than nimble, and notorious as being able to "take it." They can perhaps be looked upon as "sluggers" rather than scientific boxers. Punch-drunken states have been found in fighters of all weights, but most characteristically in small men who had not been particular about the size of their opponents, having often taken on contestants heavier than themselves. There is a correlation between the degree of cranio-facial marking and the intensity of the punch-drunk syndrome. The sum total of contests is important, as well as the number of occasions upon which the boxers have been rendered unconscious. Being "knocked out on the feet" is perhaps just as significant as being floored.

Of special aetiological importance is that humbler side of boxing where the contestant travels in fairs in the boxing-booth (or "blood-tub"), taking on all-comers at any weight. Almost as characteristic is the story of the boxer who eventually gives up the ring, having failed to make the grade, later to become what they call a "punch-bag"—that is, one of a team of sparring partners to a first-class heavyweight. A typical story is that the boxer, after a promising early career in the ring, begins to slow up; to be knocked out more often; to win fewer contests; and to be seedy for increasingly longer periods after each affray. Most characteristic of all is the admission on the part of the boxer that he finally abandoned the ring because of his wife's increasing disapproval of his career.

The symptoms and signs of a punch-drunk state are variegated, but there are certain clinical features which underlie all the diverse patterns of the disease. As a rule, gradual evolution of mental and physical anomalies marks the insidious onset of the encephalopathy. Among mental symptoms there is the slow appearance of a fatuous or euphoric dementia with emotional lability. The victim displays but little insight into his deterioration. Speech and thought become progressively slower. Memory deteriorates considerably. In Service experience there is a falling-off both in efficiency and in conduct. There may be mood-swings, intense irritability, and sometimes truculence leading to uninhibited violent behaviour. Simple fatuous cheerfulness is, however, the commonest prevailing mood, though sometimes there is depression with a paranoid colouring.

From the clinical standpoint, the neurologist may encounter almost any combination of pyramidal, extra-pyramidal, and cerebellar signs. Tremor and dysarthria are two of the commonest findings. In an earlier paper upon this topic (Critchley, 1949) three or four commonly recurring syndromes within the punch-drunk state were enumerated. Thus one could isolate a syndrome reminiscent of general paralysis; another which closely resembled disseminated sclerosis; and a third where differentiation was necessary from a frontal lobe tumour. This paper was mainly based upon wartime experience. Since the war one has been impressed with the greater frequency, in civilian practice, of types of punch-drunkenness in which pallidal and striatal defects are conspicuous. Here the clinical combination of a mask-like face, slurred monotonous speech, extrapyramidal rigidity, infrequency and slowness of movement, and tremors of the arms and head constitutes a syndrome which superficially resembles a post-encephalitic Parkinsonian state.

The foregoing constitute the clinical findings in cases of punch-drunkenness. The victim, as a matter of fact, may spontaneously admit to but few disabilities, if any at all. Here, then, is evidence both of the insidious development of the encephalopathy and of the lack of awareness so characteristic of the dementing patient. He may, however, complain of such symptoms as persistent dull headache, postural dizziness, deafness, poor vision, intolerance towards alcohol, unsteady gait, and shakiness. Very occasionally periodic convulsive seizures bring the victim to the doctor. In Service medicine the punch-drunk victim is often identified quite early for other reasons—namely, in his role as a persistent offender. Delinquency therefore may be the evidence of a rapidly changing personality, associated with progressive brain damage.

Illustrative Cases of the Punch-drunk State

The following clinical notes illustrate some of the characteristics of traumatic progressive encephalopathy in boxers.

Case 1: A Punch-drunk Syndrome reminiscent of a Frontal-lobe Tumour.—A professional heavyweight who had been sparring partner to Walter Neusel, Tommy Farr, Eddie Phillips, Max Baer, Jack Hyams, and others gave his story with extreme slowness, and some dysarthria. His actual words were, "I can't think properly and I can't remember things; my speech has been getting bad for 18 months, and for the past two weeks it has been playing hell with me. I can't talk any quicker than I am now. My head goes blank at times. I get pain over my left eye nearly every morning when I wake, but if I rest it goes away, but I feel thick-headed all day long. I get dizzy but I've never lost my senses. The left ear is a bit deaf, and the sight isn't good in the left eye. I can't give my mind to reading; it makes my head worse, I just look at the illustrations."

Case 2: Incipient Punch-drunk Disability in a First-class Heavyweight.—This boxer, who was always liable to headaches after each contest, had found them more persistent in the last few months. It was noticed by others that he was becoming a little slower, and the speech somewhat thick. On the occasion of his last contest he was knocked out, and headaches thereafter became troublesome. Although the patient maintained that he felt well, he was noticeably dysarthric and slow in cerebration. There was a strong suspicion here of an early punch-drunkenness and he was advised to rest. In this view the British Boxing Board of Control concurred, but the U.S. authorities dissented. They insisted on his fulfilling his American contracts, but thereafter he rapidly deteriorated both medically and professionally, and the diagnosis of punch-drunkenness was ultimately agreed upon.

Case 3: Mild Case of Punch-drunkenness with Dementia, Euphoria, and Dysarthria.—This man, aged 61, had fought as a professional lightweight between 1910 and 1926. He became flyweight champion of Essex. Later he went to seed and took on various female boxers, including Madame Carpentier, the world's champion lady boxer. When examined he was complaining of poor powers of concentration and forgetfulness. He would lose the thread in his conversation and had to rely on written memoranda. His mood was extremely euphoric and his speech grossly dysarthric. There was a traumatic arthritis of his thumb-joint with evidence of an old fracture. The E.E.G. was flat. The patient asserted that he would never allow his children to box, and said that "it ought to be stopped."

Case 4: Isolated Convulsive Seizure in a Professional Boxer—? Epilepsy. ? Early Punch-drunkenness.—A man aged 23, who had been a professional lightweight for about four years, had been knocked out only once. Recently, while flying from South Africa, he began to vomit in the aircraft and then lost consciousness and became convulsed. Clinical examination was negative, but the E.E.G. was abnormal, with bilateral synchronous rhythmic slow-wave

discharges suggestive of idiopathic epilepsy. He was advised not to fulfil his engagement to fight the following week; but this warning was not heeded.

Case 5: Delinquency associated with Early Punch-drunkenness.—A naval rating aged 29 was sent to the medical officer with the following report from the regulating petty officer: "He has been adrift for two and a half hours . . . he has lost his respirator in a pub. When spoken to, he belched offensively and without regard. He is a general nuisance, not intelligent, a bad influence. In short, he is evil." When asked what symptoms he complained of, he hooted with laughter and said in a grossly dysarthric fashion, "I've been adrift." Inquiry revealed that he had taken up boxing at 13 and that he had turned professional. He believed he had won about three-quarters of his 300-odd fights. Latterly, after some of his longer fights, he would be groggy and complain of misty vision.

Case 6: Gross Delinquency in a possible mild case of Punch-drunkenness.—A featherweight professional boxer aged 27, at one time sparring partner to Seaman Watson, was seen in 1942. In 1937 he had been charged with inflicting grievous bodily harm, but was acquitted. He entered the Service in 1940, and promptly went adrift for 24 hours and then deserted for 14 days. After serving 90 days' detention he almost at once deserted for 21 days. Later he deserted a third time, and was convicted of theft, going to gaol for three months. Thereafter he deserted for seven weeks and married a woman he had met the day previously. He left her immediately after the ceremony and never saw her again. Since then he was charged with a fourth term of desertion. His medical state comprised: headache, poor memory, a tendency towards losing himself in familiar environments, depression, and intolerance towards alcohol.

Case 7: Recurrent Attacks of Hysterical Blindness associated with a Punch-drunk State.—A former bantam-weight champion of Wales and of the West of England aged 27 had been knocked out some 60 times in all. He gave up the ring in 1938 after a fight on account of failing eyesight and hearing. The first attacks of blindness followed a vitriol-throwing incident when he tried to rescue a prostitute from some low joint. He had met her a few days before and had, he thought, persuaded her to go straight, and had given her her fare home. Another attack of blindness took place when he narrowly escaped being run down by a lorry. He suddenly lost his eyesight once again in the naval barracks, and he was examined two weeks later in the R.N. Hospital, Chatham, showing all the dramatic characteristics of a case of hysterical blindness. His vision suddenly returned, but it disappeared once again when he received an anonymous letter impugning the fidelity of his wife.

Case 8: Striatal Type of Punch-drunk Syndrome.—This man, aged 27, began to box at the age of 16 and turned professional. He fought about 200 contests as a bantam-weight, sometimes two or three times a week. Later he appeared in boxing booths, for it was becoming increasingly obvious to him that he was deteriorating. About this time—that is, when aged 27 years—he was rejected at the recruiting office, and he realized then that his sight was poor and that his gait was unsteady. Three months later his hands became tremulous. His symptoms increased up to a point and then remained stationary. On examination he was found to be slightly forgetful. He had a nasal type of dysarthria, his face was expressionless, and he tended to dribble. He had a tremor of the outstretched hands, some increase in muscle tonus, and slight but definite ataxia. Tendon-jerks were increased and there was a right extensor response. The gait was titubant, short-steppage, and stiff.

Case 9: Cerebellar Type of Punch-drunkenness, reminiscent of Disseminated Sclerosis.—This man, aged 37, was a professional boxer between the ages of 21 and 31. Successful at first as a lightweight, he deteriorated and took on opponents out of his weight class. He did not think he came to much harm until a particular fight in the Albert Hall when—being distracted by a spectator calling out his name—he was knocked down. He got up after a short count and was floored a second time. This state of affairs went on again and again, and though he finished the fight he subsequently remembered very little of what happened. His symptoms consisted of progressively increasing tremor and clumsiness of the right upper limb. Examination revealed a slight dysarthria, bilateral intention tremor, increased tendon-jerks, but flexor plantar responses. Appropriate tests brought to light a mild degree of intellectual deterioration.

Case 10: Striato-cerebellar Type of Punch-drunkenness.—A professional welterweight, aged 27, whose record had gradually deteriorated, had been knocked out three times. In 1948 (aged 24) after losing his temper with his fiancée, he started to tremble all over. This subsided except in the left hand, where the tremor persisted with increasing amplitude. After six months it involved the head, and his leg began to drag. His articulation altered. Examination revealed a fine tremor of the head, which was held in a curious retracted posture. The speech was dysarthric and staccato. On the left side there was an unusual type of intention tremor, associated with dysdiadochokinesis. The gait showed a steppage abnormality of the left leg. Both plantar responses were extensor in type.

Case 11: Cerebellar Type of Punch-drunkenness in an Amateur Boxer.—A man aged 39, who had been four times schoolboy champion of Great Britain, began to develop tremor of the arm during the war, on which account he was invalided from the Army. His symptoms increased slightly and he showed an impassive, rather mask-like face; marked dysarthria; titubation of the head, with some hypertrophy of the muscles of the neck; incoordination of the arms and legs, more so on the left; with intention tremor and dysdiadochokinesis. Originally of superior intelligence, he later showed both an intellectual falling-off and a curious alteration in personality.

Traumatic Encephalopathy

The interval between the onset of a boxing career and the onset of punch-drunk manifestation is often difficult to gauge precisely, but it is usually a matter of years. In an earlier paper (Critchley, 1949) it was stated that in the case of 21 punch-drunk naval patients the interval between the taking-up of a boxing career and the development of neurological symptoms or signs averaged 16 years, with extremes of 6 and 40 years. Of great interest, pathological as well as practical, is the fact that this traumatic encephalopathy is a progressive condition. Once established it not only does not permit of reversibility, but it ordinarily advances steadily. This is the case even though the boxer has retired from the ring and repeated cranial traumata are at an end.

Neurologists have visualized this state of chronic traumatic encephalopathy as being based upon multiple minor cerebral contusions, possibly with initial pinpoint haemorrhages later replaced by a gliosis, cortical atrophy, and internal hydrocephalus. It is a matter of surprise that actual pathological verification should have been so dilatory in its appearance. At the time of the publication of my earlier paper on the punch-drunk syndromes—namely, in 1949—no necropsy material was available. Since that date there has been a report of a single case of punch-drunkenness with thorough pathological study (Brandenburg and Hallervorden, 1954). The outstanding findings consisted in a rich profusion of "senile" plaques scattered throughout the shrunken cortex, especially in the frontal lobes. Although the patient was only 51 years of age at the time of death, the histological picture was that of senile dementia, or, alternatively, of an Alzheimer psychosis.

Special *in vivo* investigations of cases of punch-drunkenness may indicate the existence of a cerebral atrophy. Air-studies are of particular merit here in that they may demonstrate an atrophic process, especially of the frontal lobes, with corresponding shrinkage of the white matter. Electro-encephalography has been widely practised of late in cases of punch-drunkenness. The findings have mainly revealed a non-specific type of disordered cortical rhythm, indicative merely of an atrophic process.

This particular method of investigation is of importance in that it might conceivably prove to be a more delicate index of cerebral dysfunction than clinical study. It might lend itself to a screening procedure. By submitting boxers to this test, before a contest and after a contest with or without a knock-out episode, information of value might perhaps be forthcoming. The test could furthermore be applied to experienced pugilists of all types—that is to say, those who proclaim themselves entirely fit and who display no mental or physical deterioration, as well as those who show symptoms or signs of incipient or advanced encephalopathy. A certain amount of electroencephalographic evidence is already to hand.

Although E.E.G.s were carried out on a number of my Royal Naval cases of punch-drunkenness during the war, the literature on this particular topic starts with the paper by Guillain, Sévileano, and Fandré (1948), who observed pathological E.E.G. records in two cases. Thereafter the literature contains many references to electroencephalographic data in boxers. Though of interest, and not without promise of ultimately affording diagnostic assistance, much of the available material is contradictory (Titeca, 1952; Busse and Silverman, 1952; Temmes and Huhmar, 1952; Taylor, 1953; Marquardsen, 1954; Larsson *et al.*, 1954; Kaplan and Browder, 1954; Blonstein and Clarke, 1954; Pampus and Grote, 1956).

From one's personal experience, taken in conjunction with the published evidence, the electroencephalographic aspects of the boxing problem can perhaps be summed up by saying that: (1) minor non-specific anomalies are common in the records of experienced boxers; (2) such findings are commoner and often more intense in cases of punch-drunkenness; but (3) the E.E.G. record is at the present time a less sensitive index of punch-drunkenness than is the clinical evidence, for symptoms and signs may be present at a time when the E.E.G. records are within the limits of normality; and (4) a return to a normal state in a series of E.E.G. records taken in a particular patient cannot be regarded as a *restitutio ad integrum*, whether physiological, anatomical, or clinical.

"Groggy States"

Intermediate between the knock-out (the most acute manifestation of cerebral dysfunction among boxing injuries) and the punch-drunk state (the most chronic among the disorders) is a condition which is referred to in lay terms as the "groggy state." Here, as the result of severe battering sustained during a particular contest, the victim develops a mental confusion with subsequent amnesia, together with an impairment in the speed and accuracy of the motor skill represented by the act of boxing. The affected pugilist may continue to fight, but in an "automatic fashion," to use a lay expression again. The constellation of movements needed for defence and attack takes place at a more inferior level of consciousness, without there being a vigilant awareness of each muscular movement. Unlike many motor skills—for example, driving a car, typing, playing a musical instrument—the motor pattern of a pugilist in the ring depends upon imponderable circumstances—that is, the actions of the opponent. Consequently the movements cannot be carried out with full efficiency unless awareness is intact. Each movement is much more a willed deliberate volitional act than, say, in the case of a skilled typist or pianist, who can go through a set repertory of actions while thinking and talking of something quite different.

In the groggy state this process of active attention is at fault, with resulting falling-off in the quality of performance, more especially in attack.

Even to an unpractised eye it may be obvious that the patient in a groggy state is making feeble and uncoordinated movements, that his limbs are ataxic, and that he is inefficient and vulnerable. He is, as they say, "out on his feet." At this point the referee may stop the fight. The victim may remain indisposed for the next few days, with headaches, postural dizziness, and imbalance. During this time he may be morose and taciturn, muddled regarding recent events, and perhaps tremulous and dysarthric. This condition passes, and the boxer ordinarily returns to his *status quo ante*. It is arguable, however, that he has suffered a certain cerebral affront, for one groggy state is apt to follow another with succeeding contests. The suggestion, indeed, that an accumulation of groggy states leads to a permanent punch-drunken clinical picture is not to be considered far-fetched. Perhaps, then, we could translate the layman's term "groggy state" into "remitting commotio cerebri."

Illustrative Cases of the Groggy State

The following case notes illustrate the increasing grogginess which may follow a particular contest or series of contests.

Case 12.—This man would feel groggy for days after each contest, with his eyesight getting worse temporarily each time. He had particular difficulty in judging distances. This is why he eventually packed up boxing.

Case 13.—"I seemed to go dead, and not to feel the punches. It would now take me two or three days to get over each fight." (Ex-welterweight champion of the West of England.)

Case 14: Latterly he took longer and longer to recover from each fight, because of headaches, defective memory, and impaired speech. His wife did not approve of his boxing and persuaded him to give it up. (Ex-lightweight professional boxer with punch-drunkenness in which headaches and irritability were conspicuous.)

Case 15.—A former welterweight champion of Lincolnshire, aged 30, used to recover from his contests promptly and has even boxed on two consecutive days. Latterly he would remain stiff for some days after each contest. After two years his record deteriorated, especially when he got to London, where he found the style of boxing was different, with more "in-fighting." He stopped boxing for four years, but took it up again when he joined the Navy. In his last contest he went down several times in the fourth round, but he finished the fight "not feeling right" . . . his "limbs felt like rubber" and he complained of a thick head and a sore neck, and he could not sleep. Early morning headaches followed, with severe postural giddiness, irritability, tearfulness, poor memory, fatigue, and lack of interest. Examination brought out that his emotivity and a gross dysarthria were evident.

Case 16.—A man aged 29, never quick on his feet, he could always "take it," and ordinarily "did twelves" (as he put it when questioned on the length of the contests he fought). He packed it up because of his headaches. After one fight he became so unsteady on his legs that he was accused of being drunk. Towards the end of his boxing career he would feel queer for about a week after each fight.

Case 17.—A man aged 28; two weeks before entering hospital he staggered so much in the ring that the audience howled him down and the promoter declared him drunk. In hospital he presented a picture somewhat resembling disseminated sclerosis. Three weeks after his discharge from hospital he fought Paolino in Paris for the heavyweight championship of Europe (this was in 1932), but he was knocked out at the first blow and the crowd stormed the ring.

Case 18.—This man, aged 25, had been four times middleweight champion of Wales and sparring partner to Jack Petersen and to Len Harvey. About six months before coming into hospital he received some blows from Jack Petersen. He had to fight again two days later, but gave up after three rounds because of weakness in his arms. His next fight was a fortnight later; he gave up after the second round because of weakness and tremulousness of his limbs. His manager thought he was drunk. A doctor told him never to box again. He seemed to improve after a month's convalescence in Swansea, but, still being unsteady, he went into the National Hospital, where he was found to be punch-drunk.

Case 19.—A heavyweight aged 31 was knocked out "through the ropes" in 1945 and was dazed for the next 24 hours. The right leg was found to be weak immediately after his fight and continued so. Within the day or two following this same fight his right hand began to shake: the left hand started to tremble some six months later. He was hospitalized seven years later, a typical severe punch-drunk.

Case 20.—A delinquent mild punch-drunk, interviewed in prison, gave up boxing at the age of 20 because he was not feeling himself. He felt drunk although he had taken nothing in the way of alcohol: his head would be in a swim after each fight.

Case 21.—A Royal Navy welterweight, aged 22, matched against a tougher opponent, was struck heavily over the heart in the second round, causing him to feel dizzy or groggy. Shortly after he got a severe blow to the side of the head, flooring him for a count of nine, when he rose and resumed the fight. He could remember vividly every incident up till then; but after that his memory was a blank. Apparently he finished the second round, and in the third round he received heavy punishment, being knocked down 10 times, striking his head on the floor more than once. His next recollection is being on the deck of his ship about an hour later, feeling dizzy and sore about the stomach. Two weeks later he became ill with headaches, vertigo, and vomiting, and was hospitalized. Investigation showed old blood-staining of the C.S.F.

Conclusions

There is much in boxing to interest a practising neurologist, and special attention should be focused upon (1) the phenomenon of groggy states as occurring during or after a contest, and (2) the condition known as traumatic progressive encephalopathy (or punch-drunkenness).

Owing to the extreme paucity of pathological data, it is highly desirable that opportunity should be afforded neuropathologists of studying the appearances in the brain of punch-drunk patients.

There is a scope for further study of boxers by electroencephalographic as well as clinical techniques, at all stages: that is, early as well as late in their careers; and before and immediately after a contest, especially if a knock-out has occurred.

For permission to quote the naval cases I am grateful to the Medical Director-General of the Royal Navy.

REFERENCES

Blonstein, J. L., and Clarke, E. (1954). *British Medical Journal*, **2**, 1523.
Boisey M. (1938). *Lésions et traumatismes sportifs*. Paris.
Brandenburg, W., and Hallervorden, J. (1954). *Virchows Arch. path. Anat.*, **325**, 680.
Busse, E. W., and Silverman, A. J. (1952). *J. Amer. med. Ass.*, **149**, 1522.
Critchley, M. (1949). "Punch-drunk syndromes: the chronic traumatic encephalopathy of boxers," from *Hommage à Clovis Vincent*. Maloine, Paris.
Denny-Brown, D., and Russell, W. R. (1941). *Brain*, **64**, 93.
Doggart, J. H. (1951). *Trans. Ophthal. Soc. U.K.*, **71**, 53.
—— (1955). *A.M.A. Arch. Ophthal.*, **54**, 161.
Favory, A., and Sédan, J. (1951). *Arch. Ophtal.*, **11**, 429.
Flint, O. (1920). *Boxer*. Leipzig and Zürich.
Guillain, G., Sévilcano, E., and Fandré, M. (1948). *Bull. Acad. nat. Méd. (Paris)*, **132**, 394.
Goltz, F. (1863). *Virchows Arch. path. Anat.*, **24**, 1.
Gonzales, T. A., Vance, M., and Helpen, N. M. (1937). *Legal Medicine and Toxicology*. Appleton-Century Co., New York.
Hering, H. E. (1927). *Die Karotissinusreflexe auf Herz und Gefässe*. Dresden and Leipzig.
Johansen, O. (1955). *Idrett og skader*. Oslo.
Jokl, E. (1941). *The Medical Aspects of Boxing*. Van Schaik, Pretoria.
Kaplan, H. A., and Browder, J. (1954). *J. Amer. med. Ass.*, **156**, 1138.
La Cava, G. (1949). *Bruz.-méd.*, **29**, 3233, 3304.
—— (1952). Ibid., **32**, 61, 120.
Larsson, L. E., Melin, K. A., Nordström-Ohrberg, G., Silfverskiöld, B. P., and Ohrberg, K. (1954). *Acta psychiat. neurol. scand.*, Suppl. 95.
Marquardsen, J. (1954). Ibid., **29**, 30.
Martland, H. S. (1928). *J. Amer. med. Ass.*, **91**, 1103.
Pampus, F., and Grote, W. (1956). *Arch. Psychiat. Nervenkr.*, **194**, 152.
Pfeiffer, K. (1922). *Der Faustkampf*. Leipzig.
Sherrington, C. (1906). *Integrative Action of the Nervous System*. London, Somen, H. (1913-14). *Paris méd.*, **13**, 54.
Steinhaus, A. H. (1950a). *Look*, **14**, 35.
—— (1950b). *Amer. Forum of the Air*, **13**, 3.
—— (1951). *J. Amer. Ass. Hlth.*, **22**, No. 8, 12.
Taylor, R. B. (1953). *British Medical Journal*, **1**, 200.
Temmes, Y., and Huhmar, E. (1952). *Acta psychiat. neurol. scand.*, **27**, 175.
Titeca, J. (1952). *Bull. Acad. Méd. Belg.*, **17**, 507.
Winterstein, C. E. (1938). *Med. Wld (Lond.)*, **49**, 427.

FURTHER OBSERVATIONS ON THE MEDICAL ASPECTS OF AMATEUR BOXING

BY

J. L. BLONSTEIN, M.R.C.S., L.R.C.P.
Honorary Medical Officer to the Amateur Boxing Association

AND

EDWIN CLARKE, M.D., M.R.C.P.
Lecturer in Neurology, Postgraduate Medical School of London

The medical aspects of boxing continue to attract attention in both the medical and the lay press. Recent attacks on the sport (Doggart, 1955; Summerskill, 1956) have aroused further controversy, and the reports of death (Rottgen, 1955; Pampus and Müller, 1956; Müller, 1956) and of possible brain damage (Taylor, 1953; Geller, 1953; La Cava, 1954; Brandenburg and Hallervorden, 1954; Soeder and Arndt, 1954; Pampus and Grote, 1956) repeatedly raise the question of the advisability of allowing youths to indulge in what would appear to be a dangerous and unjustifiable recreation. We therefore thought it worth while to review the boxing injuries, especially those to the head, encountered in the London Amateur Boxing Association contests during the past season, as an extension of a previous report (Blonstein and Clarke, 1954).

Material

During the period of seven months (October, 1955, to April, 1956, inclusive) there were approximately 300 tournaments representing about 3,000 boxing contests under the control of the London Amateur Boxing Association, and these involved almost 5,000 boxers. According to the medical welfare scheme instituted two years ago (Blonstein and Clarke, 1954), all contestants who have been concussed or have suffered lengthy amnesia are rested for at least one month, the exact period depending upon the estimated severity of the injury. It was therefore possible to trace with ease all the boxers who had been knocked out during the season. Those who had been more severely concussed or who had been knocked out more than once were selected for special study. There were 29, and their ages ranged from 17 to 26 years; 23 had been knocked out twice and 6 had been severely concussed on one occasion.

Method

First an attempt was made to classify the boxers according to the severity of their injury, as judged by events in the ring. The following groups could be identified.

Group I: "Out on the Feet."—Following a head blow the boxer was unable to defend himself, was obviously dazed, and staggered about the ring or rested on the ropes.

Group II: Mild Knock-out.—Those who were knocked down and could not rise before the count of 10, but who, although dazed, were not unconscious for more than a few seconds.

Group III: Moderately Severe Knock-out.—Those rendered unconscious, and carried to their corner, where they quickly recovered.

Group IV: Severe Knock-out.—Those remaining unconscious for periods greater than Group III.

This classification accounted for only 26 of the 29 cases, for at times there was a disparity between the observed actions of boxers and the amnesia estimated subsequently.

As the behaviour of the boxer in the ring is obviously of considerable importance, a grouping based on this rather than total traumatic amnesia alone was selected. Both, however, were recorded in every instance.

Each of the 29 boxers was given a careful neurological examination after the responsible contests, and an electroencephalogram (E.E.G.) was carried out. Seven had the E.E.G. within two weeks of the knock-out and 22 had it three to four weeks afterwards. The difficulties in arranging this examination for amateur boxers have been discussed previously (Blonstein and Clarke, 1954); a recording immediately after a bout was never practicable.

Results

The 29 boxers represented 0.58% of all boxers active during the 1955–6 season, so that, roughly speaking, in each 100 contests there was one such person involved. Their distribution according to weight was: heavyweight, 4; light heavyweight, 7; middleweight, 5; light middleweight, 4; welterweight, 2; light welterweight, 3; lightweight, 1; featherweight, 1; bantamweight, 1; flyweight, 1.

As would be expected, the boxer's weight varied directly with the frequency and severity of knock-outs. The grouping relative to the severity of the knock-out in 26 cases was: group I, 6 cases; group II, 7 cases; group III, 7 cases; and group IV, 6 cases.

In these youths the central nervous system was normal when examined soon after the head injury and no E.E.G. abnormalities were detected in any of them.

The three cases that could not be included in the suggested classification are described individually because they illustrate some of the varieties of memory loss encountered.

Amnesia may occur without an obvious knock-out. One boxer completed his bout and was given the decision on points, but in the dressing-room he complained of headache and had amnesia for the whole contest and its sequelae. On examination the left pupil was larger than the right, but the nervous system was otherwise normal. He had received several blows to the jaw, but had not fallen, and his condition during the contest had given rise to no comments or anxiety, despite the fact that he had no memory of events in the ring. His E.E.G. was normal, but he was not allowed to box for one month. This case points out the disparity that may exist between the boxer's behaviour and his subsequent amnesia.

Sometimes the head blows may be trivial but the combined amnesia (retrograde and post-traumatic) may be disproportionately large. In the second case, a youth who was unconscious for only a few seconds during his contest was confused and forgetful the next day. Instead of going to his usual place of work he travelled to a different town. The nervous system and E.E.G. were normal. There is reason to believe that his lengthy amnesia was in part "functional" in origin.

A delayed loss of consciousness has been observed, and in the following case it closely resembles that described by Denny-Brown (1941). A boy was "out on his feet" at the end of the first round and the contest was stopped. He complained of double vision on looking to the left, but had a shower and his refreshments. About half an hour later he became unconscious and was removed to hospital, where he regained consciousness 12 hours later. There were no abnormal neurological signs and the E.E.G. was normal. This boy made a complete recovery, but has been advised to give up boxing permanently.

The E.E.G. was therefore normal in each of the 29 boxers and the neurological abnormalities were extremely few. If loss of memory alone in the 26 cases is considered without reference to the suggested grouping, three youths had retrograde amnesia of up to one hour and in seven the post-traumatic amnesia lasted from half an hour to three hours. The maximum period of unconsciousness, however, was half an hour.

During the 1955–6 season there were no skull fractures, and injuries elsewhere in the body were few. One boxer suffered a spontaneous pneumothorax, another fractured a fibula, and there were 11 cases of fractured metacarpals. Cuts of the supraorbital ridges and cheeks occurred approximately twice in every tournament; not a single case of fractured nose or serious eye injury has been reported.

Discussion

Medical control of boxing, both amateur and professional, is of vital importance if the sport is to continue, and recent reports indicate the prophylactic measures used or suggested in various countries (Labignette, 1954, in France; Egan, 1954, in the U.S.A. (see also Ferlaino, 1952); Schwarz, 1955, in East Germany; Henner, 1955, in Czechoslovakia; Graham, 1955, in Great Britain; Medved, 1955, in Yugoslavia). We are concerned here with the amateur boxer only, and it is obvious that youths at school, at work, or in the armed Forces who are indulging in the sport as a pastime rather than as a profession must be given the maximum of protection; an impeccable medical service is therefore mandatory.

We have described elsewhere the scheme adopted by the London Amateur Boxing Association (Blonstein and Clarke, 1954), and it would appear from a survey of a season's boxing that damage to the brain, claimed to be severe by most opponents of the sport, may not be a serious objection when medical supervision is adequate. By clinical and electroencephalographic means we have detected no acute or subacute disabilities resulting from boxing blows to the head, and injuries elsewhere in the body on the whole have been few. A survey of casualties in other sport over a similar period of time would be of interest; it seems that in New York, for example, it is more dangerous to play baseball or football than to box (Gonzales, 1951).

That deleterious cerebral changes may occur in amateurs after an interval is a possibility that has by no means been excluded by this study. There have been allegations that it is inevitable; but there is in fact very little, if any, evidence to support them. There is probably more to indicate the reverse. Thus in our experience the "punch-drunk" syndrome has never been observed in amateur boxers (Blonstein and Clarke, 1954), and, furthermore, there are many men in the various professions who, although keen amateur boxers in their youth, have demonstrated by their careers that a cerebral deficit has not ensued. However, a follow-up over a long period of time has not yet been carried out in either professional or amateur boxers, so that the precise frequency of the "punch-drunk" state is unknown. On the whole it seems that the much-publicized pugilist's dementia is in amateur boxers as unlikely as the deleterious effects of "athlete's heart" in non-professional athletes.

The popular concept that each major head blow produces a petechial haemorrhage in the boxer's brain has been fostered by Martland (1928), who was one of the first medical observers to describe the "punch-drunk" syndrome. But, as Kaplan and Browder (1954) have pointed out, there is no pathological verification of this. Evidence from animal investigations (Windle, Groat, and Fox, 1944), however, suggests that a blow sufficient to induce unconsciousness, as as well as heavy blows that do not, will produce histological changes in the form of cell death. It follows that if these changes take place in the human brain, repeated stimuli of the same nature will eventually bring about clinically detectable brain degeneration such as that seen in the professional pugilist after a long and misguided career (Brandenburg and Hallervorden, 1954); that a very severe closed head injury in man will produce white-matter degeneration has recently been shown by Strich (1956).

So far as the present considerations are concerned there are two objections to this assumption. In the first place, the juvenile brain, like any other young organ, can presumably adapt itself to the small amount of cell loss, if this in fact takes place. The second point is the more

important, because if there is a strict medical control of amateur boxers the possibility of extensive and repeated neuronal destruction will not exist and the argument indicting boxing on account of the cerebral end-results will therefore be groundless and unnecessary. Thus according to the London Amateur Boxing Association laws, boxers who are repeatedly knocked out, who suffer lengthy amnesias on several occasions, or who are severely concussed are excluded from further contests.

There is another aspect of the medical service, and this is to explore all possible measures that will help to safeguard the non-professional boxer. As well as our report on the use of the E.E.G. as a prophylactic aid, other authors have published their results (Ardito, 1954; Soeder and Arndt, 1954; Larsson *et al.*, 1954; Medved, 1955; Pampus and Grote, 1956). Kaplan and Browder (1954) made an extensive study of professional boxers, and they, like most other authors and ourselves, found little or no evidence of permanent brain changes. Records taken soon after a bout in which the boxer received a lot of head blows were usually normal, and a few men who were followed for up to three years whilst boxing regularly developed no E.E.G. abnormality. Permanent cerebral deficits, although they no doubt occur, seem to be very difficult to detect by the methods of investigation at present available, and are in any case probably rare. Kaplan and Browder (1954), when evaluating the relative merits of the information obtained from these investigations, arrange them in the following order of usefulness: past medical history, physical fitness, past boxing performances, and finally the E.E.G. With this we would agree, and it is obvious that a decision concerning a boy's boxing future can be reached only by considering all the available evidence.

At the moment, all the possible information concerning boxing head blows must be amassed, and observations on the amnesia of the concussed boxer is of considerable importance. The retrograde and post-traumatic memory losses are both indications of the severity of the blow (Russell and Nathan, 1946), but they must be assessed with reference to events observed by onlookers. Thus, a lengthy amnesia without the external appearances of loss of consciousness should be considered equivalent to a state where the boxer is obviously unconscious.

The grading of head blows received in the ring is not easy, and we have attempted to provide a classification that seems to include most cases of concussion. It is possible that as we acquire more knowledge some modification may be necessary, but at the moment the recording of both objective and subjective information is justified.

Much more information on boxing amnesia, however, is required, and, as well as the immediate retrograde type, the behaviour of more distant pre-traumatic memories may be important (Williams and Zangwill, 1952). It seems possible that assessment of the total traumatic amnesia will be more important than the neurological examination, the E.E.G., or other tests in detecting the first signs of the deleterious effects of boxing upon the brain—if such will ever result from amateur boxing.

The increase of medical interest in amateur boxing that is evident all over the world is of considerable importance. It is helping to eradicate the prejudices and misconceptions that dominate most discussions on the subject. At the same time, it will improve and popularize the medical welfare of youths who indulge in boxing as a recreation and in particular will protect them from chronic cerebral damage, if this can be proved to be a consequence of amateur boxing. The medical problems of the amateurs are under constant review and new knowledge acquired from the sport or from elsewhere will be made use of where appropriate.

With regard to the more fundamental objections to boxing as a sport, we have nothing to add to previous statements (Blonstein and Clarke, 1954), except to comment upon the interesting fact that the emotions said to be displayed in the ring and condemned by the opponents of boxing are often evident in their belligerent verbosity.

Summary

Boxing injuries encountered in 3,000 London Amateur Boxing Association contests during 1955–6 are assessed.

Only 29 boxers (0.58%) were severely concussed or knocked out more than once. The results of neurological and electroencephalographic examinations were normal in all. Other injuries were very few.

A classification of boxing head injuries is suggested and the importance of traumatic amnesia stressed.

Doubt is cast upon the possibility of chronic cerebral changes in amateur boxers who are protected by an adequate medical service.

In view of the limitations of clinical, electroencephalographic, and other investigations the evaluation of memory loss may be of great importance as a prophylactic aid.

REFERENCES

Ardito, R. (1954). *Riv. Neurol.*, **24**, 731.
Blonstein, J. L., and Clarke, E. (1954). *British Medical Journal*, **2**, 1523.
Brandenburg, W., and Hallervorden, J. (1954). *Virchows Arch. path. Anat.*, **325**, 680.
Denny-Brown, D. (1941). *Lancet*, **1**, 371.
Doggart, J. H. (1955). *A.M.A. Arch. Ophthal.*, **54**, 161.
Egan, D. J. (1954). *Med. Techn. Bull.*, **5**, 102.
Ferlaino, F. R. (1952). *J. Amer. med. Ass.*, **150**, 651.
Geller, W. (1953). *Nervenarzt*, **24**, 69.
Gonzales, T. A. (1951). *J. Amer. med. Ass.*, **146**, 1506.
Graham, J. W. (1955). *British Medical Journal*, **1**, 219.
Henner, K. (1955). *Cas. Lék. čes.*, **94**, 859.
Kaplan, H. A., and Browder, J. (1954). *J. Amer. med. Ass.*, **156**, 1138.
Labignette, P. (1954). *Concours méd.*, **76**, 4204.
La Cava, G. (1954). *Dtsch. med. Wschr.*, **79**, 817.
Larsson, L. E., Melin, K. A., Nordström-Öhrberg, G., Silfverskiöld, B. P., and Öhrberg, K. (1954). *Acta psychiat. neurol. scand.*, Suppl. 95.
Martland, H. S. (1928). *J. Amer. med. Ass.*, **91**, 1103.
Medved, R. (1955). *Dtsch. med. Wschr.*, **80**, 191.
Müller, N. (1956). *Dtsch. Z. ges. gericht. Méd.*, **44**, 763.
Pampus, F., and Grote, W. (1956). *Arch. Psychiat. Nervenkr.*, **194**, 152.
—— and Müller, N. (1956). *Dtsch. Z. Nervenheilk.*, **174**, 177.
Rottgen, P. (1955). *Med. esp.*, **34**, 35.
Russell, W. R., and Nathan, P. W. (1946). *Brain*, **69**, 280.
Schwarz, B. (1955). *Dtsch. GesendhWes.*, **10**, 1319.
Soeder, M., and Arndt, T. (1954). *Dtsch. med. Wschr.*, **79**, 1792.
Strich, S. J. (1956). *J. Neurol. Neurosurg. Psychiat.*, **19**, 163.
Summerskill, E. (1956). *The Ignoble Art*. Heinemann, London.
Taylor, R. B. (1953). *British Medical Journal*, **1**, 209.
Williams, M., and Zangwill, O. L. (1952). *J. Neurol. Neurosurg. Psychiat.*, **15**, 54.
Windle, W. F., Groat, R. A., and Fox, C. A. (1944). *Surg. Gynec. Obstet.*, **79**, 561.

A FATAL INJURY AT BOXING (TRAUMATIC DECEREBRATE RIGIDITY)

BY

MILROY PAUL, M.S., F.R.C.S.

Professor of Surgery, University of Ceylon

A fatal outcome from a punch on the head at boxing is fortunately a rare event, and when it does occur a sportsman who was fighting fit has been lost. The clinical and post-mortem findings in these fatal cases provide a continuous set of observations of the victim from before the beginning of the contest to the post-mortem examination. As the objective of a combatant at boxing is to punch the opponent on the head with sufficient force to drop him to the mat and cause him to remain there till he is counted out by the referee, it is evident that the victim of a "knock-out" drops because he has suffered a concussion of the brain. It is expected that the concussion will last just long enough for the count, and that recovery will be full and complete after this.

In the occasional fatal case the questions arise: has the victim died of concussion? and has he sustained additional injuries to the brain which caused his death? These are questions which could be decided by a post-mortem examination. The fatal accident also provides

the opportunity for investigating the mechanics of a punch delivered by a gloved hand on the head of an opponent who was prepared for the attack, in which the direction and the force of the punch are observed, and the condition of the victim is observed before, at the time of, and after the blow. If concussion be the cause of death, here is a complete picture of the condition. If intracranial haemorrhages have resulted from the blow, the part that these play in determining the fatal outcome can be decided. These features were well presented in the case under review.

Case History

On June 21, 1956, at an amateur boxing meeting open to boxers who had not won a trophy at an intermediate or open meeting, two entrants met for a bantam-weight—118 lb. (53.5 kg.) and under—contest. The victim of the "knock-out," Raymond Perera, was 114 lb. (51.7 kg.) and his opponent 116 lb. (52.6 kg.). Both contestants were 20 years old. The judge, Mr. A. B. Henricus, gave me his observations. The knock-out was delivered shortly after the first minute of the first round. Up to this stage the deceased, Perera, had been leading "lefts" at his opponent and the blow which knocked him out was the first struck by his opponent. It was a right cross which caught Perera on the left side of his chin. Perera appeared to be stunned by the impact, and gradually went dizzier, and slowly slumped down till his bottom reached the canvas. Then he rolled over to one side. He did not appear to be unconscious, and he was raised by his two seconds and helped to walk to his corner.

Mr. A. I. Obeysekera, who was the time-keeper at this meeting, was watching this match between rounds. He observed that when Perera reached his chair he was lying with his limbs fully extended and his jaws clenched, and that he was breathing deeply and audibly like a man sound asleep. To Mr. Obeysekera these appearances were unreal and so unlike those of the victim of a knock-out that he wondered whether the victim was shamming. He had noticed that the victim had never spoken from the time of the knock-out and that he still remained rigidly extended when being lifted into the stretcher for transport to hospital. The knock-out had occurred at 6.30 p.m. I was on duty at 7 p.m. in the casualty ward of the General Hospital, Colombo, when Perera was brought in. He lay on his stretcher breathing deeply as if in deep sleep, his lower limbs were rigidly extended at the hips and knees, and his upper limbs lay by the side of the body rigidly extended at the elbows, with the forearms fully pronated and the fists clenched. Passive movements were resisted by the rigidity, and if the resistance was overcome by force there was movement of the limb, restoring it to its original position. Perera was unconscious, and made no response to a pin tickling the inside of the nostril or pricking the skin of an eyelid.

The left pupil was widely dilated, the right pupil was small. There were no external injuries, no bruises, and no haemorrhage from the nostrils, mouth, or chin. The temperature was 97.4° F. (36.3° C.), pulse full and regular 104, and respirations 20.

At 8 p.m. Mr. C. P. De Fonseka made a trephine hole in the left temporal fossa, and found the dura mater bulged with subdural blood. Incision of the dura mater evacuated 2 oz. (57 ml.) of fluid blood. A second trephine hole in the right temporal fossa showed no extradural or subdural blood. At 11 p.m. the pulse was 88, respirations 38, and temperature 102° F. (38.9° C.). Perera's condition continued to deteriorate and he died at 1.30 a.m. on June 22.

I obtained permission of the coroner to make a post-mortem examination on June 22 at 11 a.m. On turning down the scalp there were diffuse films of coagulated blood on the subepicranial surface of the scalp and on the pericranial surface of the skull extending upwards from the temporal fossae from the surgical incisions in the scalp.

When the vault of the skull was removed, the dura mater covering the whole of the convex surface of the left cerebral hemisphere was blue from subdural blood, and when the dura mater was incised a thin film of fluid subdural blood covering the convex surface of the left cerebral hemisphere flowed away. The blood was pooled posteriorly, where it bathed the left occipital lobe. The brain was not compressed by the blood. There was no subdural blood over the right cerebral hemisphere. There was a film of subarachnoid blood coating the pre- and post-central convolutions of both cerebral hemispheres extending a little in front of and behind these convolutions as well.

On the left side this film of subarachnoid blood continued into the Sylvian fissure over the island of Reil, and over the outer surface of the left temporal lobe. The medial and inferior surfaces of the cerebral hemispheres had no films of subarachnoid blood. There were no cerebral lacerations. There was about 3 oz. (85 ml.) of dark blood in the posterior fossa, but the cerebellum and the brain stem showed no subarachnoid films of blood and no lacerations, and it is likely that this blood had flowed into the posterior fossa from above.

Sections of the cerebral hemispheres showed no intracerebral haemorrhages or lacerations, but the white matter of both hemispheres was studded with the pinhead orifices of dilated vessels. There were no haemorrhages at these orifices. Sections of the brain stem and the cerebellum showed no haemorrhage or laceration at these sites.

Discussion

The decerebrate rigidity of the victim of this bout presented some striking features. That it came on with the receipt of the blow was evident from the observation that the victim appeared to be stunned and to get dizzier, and that he then slowly slumped down till his bottom touched the canvas, an unusual fall for a knock-out blow which would be explicable on the basis that the decerebrate rigidity had broken the fall. Then, too, the "automatic walk" back to the corner, when he had been picked up and supported by his two seconds, is another phenomenon referable to the state of decerebrate rigidity. The poker-back posture when he was laid down was clear evidence of the state of decerebrate rigidity, as was the maintenance of this posture till the time of death. Experimental transections of the mid-brain have established that decerebrate rigidity is the result of a lesion between the vestibular and the red nuclei. The post-mortem examination showed that there was no visible lesion in the mid-brain, but it did establish that there had been an appreciable displacement of the cerebral hemispheres within the brain case. The subarachnoid haemorrhages on the convex surfaces of both cerebral hemispheres across their waists between their frontal and occipital poles is clearly the result of an upward drive of both cerebral hemispheres against the cranial vault from the blow on the chin being transmitted upwards through the condyles of the mandible. The diffuse subdural blood covering the convex and inferior surfaces of the left cerebral hemispheres must have been from a traction rupture of veins in the subdural space passing from the dura mater to the inferior surface and the lower convex surface of the left cerebral hemisphere. As the punch was delivered on the left of the chin this would account for the greater upward shift of the left cerebral hemisphere rupturing the veins in the subdural space on this side.

The post-mortem findings afford clear evidence of an upward shift of both cerebral hemispheres, and it would be logical to deduce that this had determined the strain in the mid-brain between the vestibular and the red nuclei which had caused the condition of decerebrate rigidity.

In this case the site of the strain in the mid-brain is postulated with precision because of its causing the condition of decerebrate rigidity. The theory that concussion is the result of a strain in the mid-brain just above the red nuclei cannot be postulated with the same certainty, but the evidence of this case in which the strain occurred at a slightly lower level is good evidence for the mid-brain lesion of concussion.

Brain Injuries at Boxing

Boxing is a well-established sport, and the rarity of fatal accidents at boxing and the absence of apparent ill effects in combatants encourage its continuance. Although the head may well receive many punches during a bout, the brain escapes damage. What is the reason for this? It must be that the positioning and tensing of the head and neck, when it is about to receive a punch, prevents the brain from being displaced. In the occasional instance where this protective position is ineffective, the brain does shift within the brain case, and the effects of this are to be assessed by examining the fatal case. The present case details such an examination. The evidence in this case permitted a reconstruction of the mechanics of the injury, and this reconstruction throws light on the mechanics of concussion.

Summary

The case is described of a boxer aged 20 who was knocked out by a blow on the chin and died in consequence. Between the knock-out and the intracranial operation the patient was in decerebrate rigidity.

BIBLIOGRAPHY

Blonstein, J. L., and Clark, E. (1954). *British Medical Journal*, 2, 1523.
Laurie, J. C. (1955). Ibid., 1, 227.
Winterstein, C. E. (1937). *Lancet*, 2, 719.

ANTIGENIC ACTIVITY OF BRITISH POLIOMYELITIS VACCINE

A REPORT TO THE COMMITTEE ON LABORATORY INVESTIGATIONS OF POLIOMYELITIS OF THE MEDICAL RESEARCH COUNCIL*

During May and June, 1956, three batches of poliomyelitis vaccine were used for the inoculation of 200,000 children in Britain under the scheme organized by the Ministry of Health. The antigenic activity of these three batches was studied in a group of children who were shown to have no detectable antibodies to any of the three types of poliomyelitis virus before their course of inoculations. The group consisted of 196 children between the ages of 1 and 11 years, in five different areas —London, Manchester, Central Scotland, Sheffield, and Southend-on-Sea.

Each batch of vaccine was also tested for antigenic activity in monkeys.

Vaccine

The three batches—designated 4, 5, and 6—were prepared by the Glaxo Laboratories under the direction of Dr. W. Wood from virus strains Brunenders (type 1), MEF-1 (type 2), and Saukett (type 3) by the formalin-inactivation method introduced by Salk. The vaccine differed in its type 1 component from that produced in America, the Mahoney strain having been replaced by a partially attenuated variant of the Brunhilde strain (Enders *et al.*, 1952; Sabin, 1955). Gear (1956) has called this the Brunenders strain.

Titration of Sera

Serial dilutions of serum in Hanks basic salt solution were mixed with an equal volume (0.25 ml.) of virus suspension in Parker 199 solution containing approximately 50 $TCID_{50}$ of the required type of virus. The mixtures were held at 37° C. for three hours, and then added to tubes containing 6- to 8-day-old monkey kidney cell cultures; two tubes were used for each serum dilution. The inoculated tubes were incubated at 37° C. in roller drums for six days, when the final readings of the cytopathic effect of virus were made and the titre (PD_{50}) of the serum determined. Titres are given as the dilution of serum in the serum–virus mixtures, as recommended by the Committee on Laboratory Investigations of Poliomyelitis.

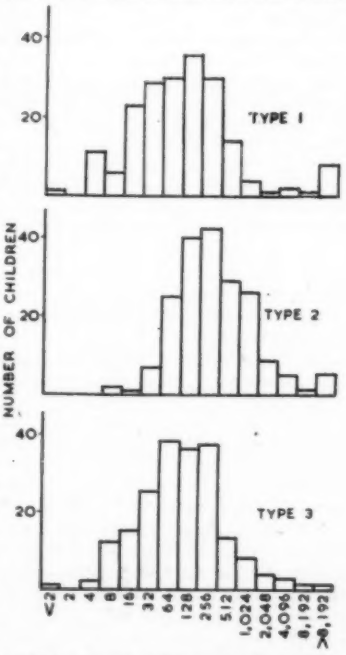
All sera were titrated, using Brunenders, MEF-1, and Saukett as the test viruses. Twenty post-immunization sera were also titrated for type 1 antibody, using Mahoney as the test virus. They gave similar results to those obtained with Brunenders, the geometric mean titres being 1/90 with Brunenders and 1/70 with Mahoney.

As controls the three British Standard poliomyelitis antisera (to be published) were always titrated in parallel in each set of tests and gave consistent results throughout. Each standard serum has been assigned a potency of 10 units per ml. and, by the method of titration employed in this study, gave on the average a titre of 1/480, 1/806, and 1/960 for types 1, 2, and 3 respectively.

Antigenic Activity in Children

Of the 196 children selected for the study, the majority had their pre-immunization sera taken on the day the first dose of vaccine was given and the remainder within a month before the first dose. The sera were shown to be negative at a titre of 1/8 to all three types of poliomyelitis virus. Seventy-two of the children were injected with batch 4, 66 with batch 5, 53 with batch 6, and 5 with a mixture of batches. Each child received two injections, each of 1 ml., at an interval of three to four weeks. The injections were given intramuscularly into the left deltoid; no untoward reactions were observed in any of the children. Serum was taken on the average two weeks after the second dose and titrated for antibody to each of the three types. Those sera showing no antibody at 1/8 were retested at 1/2 and 1/4.

Table I gives the results of the three batches of vaccine together in each area and in all areas combined; the combined results are also given in the figure. The results clearly show that almost all children responded; of the 190 whose sera were fully examined, all except two responded to all three types. It is noted that the distribution of type I antibody titres in Manchester differed from that in the other areas; of the 33 sera from Manchester, 9



Antibody titres in children after immunization with poliomyelitis vaccine.

Children were immunized and sera collected by Dr. A. Dudgeon, Dr. J. C. W. MacFarlane, Dr. D. O'Brien, and Dr. A. M. Peach (London); Dr. P. B. Stones and the medical staff of the Health Department (Manchester); Dr. N. R. Grist, Dr. W. M. Jamieson, Dr. R. C. McLeod, Dr. B. R. Nisbet, and Dr. E. Neil Reid (Scotland); Dr. J. Rendle-Short, Professor C. H. Stuart-Harris, and Dr. D. Tyrrell (Sheffield); Dr. J. Greenhalgh, Dr. J. Stevenson Logan, Dr. A. D. Macrae, Dr. Gladys Neill, Dr. R. Pilsworth, and Dr. J. C. Preston (Southend-on-Sea).

Laboratory investigations were made by Miss A. J. Ferguson, Dr. F. T. Perkins, Mrs. K. Russell, and Dr. J. O'H. Tobin, of the Biological Standards Control Laboratory, Hampstead.

The results were analysed and the report prepared by Dr. F. T. Perkins and Dr. J. O'H. Tobin

TABLE I.—*Antibody Titres in Children after Immunization with Poliomyelitis Vaccine*

Area	No. of Sera	Virus Type	Number of Sera with Antibody Titre of:															Geometric Mean Titre
			<2	2	4	8	16	32	64	128	256	512	1,024	2,048	4,096	8,192	>8,192	
London	31	1	1	0	3	0	5	6	3	5	5	0	1	1	1	0	0	60
	30	2	0	0	0	0	0	2	4	10	6	3	2	0	1	0	2	250
	31	3	0	0	0	0	2	6	9	3	3	2	2	0	2	1	1	157
Central Scotland	53	1	0	0	4	4	9	5	10	7	9	4	0	0	1	0	0	58
	48	2	0	0	0	1	1	2	10	11	13	4	5	1	0	0	0	168
	53	3	1	0	0	2	8	4	13	9	10	2	2	2	0	0	0	86
Manchester	33	1	0	0	2	0	2	1	5	4	5	3	2	0	0	1	8	144*
	33	2	0	0	0	1	0	1	5	9	5	7	2	0	2	0	1	246
	33	3	0	0	1	4	2	1	6	8	6	1	2	1	1	0	0	100
Sheffield	25	1	0	0	2	0	1	6	4	4	5	2	1	0	0	0	0	80
	25	2	0	0	0	0	0	0	2	4	8	3	3	4	0	1	0	420
	25	3	0	0	0	1	2	1	2	6	9	3	0	1	0	0	0	146
Southend-on-Sea	54	1	0	0	0	2	6	11	8	16	6	5	0	0	0	0	0	77
	54	2	0	0	0	0	0	2	4	6	10	12	14	4	2	0	0	428
	54	3	0	0	1	5	1	13	8	10	9	5	2	0	0	0	0	80
All areas	196	1	1	0	11	6	23	29	30	36	30	14	4	1	2	1	8	72*
	190†	2	0	0	0	2	1	7	25	40	42	29	26	9	5	1	3	282
	196	3	1	0	2	12	15	25	38	36	37	13	8	4	3	1	1	101

TABLE II.—*Antibody Titres in Children after Immunization with Three Different Batches of Poliomyelitis Vaccine*

Vaccine Batch	No. of Sera	Virus Type	Number of Sera with Antibody Titre of:															Geometric Mean Titre
			<2	2	4	8	16	32	64	128	256	512	1,024	2,048	4,096	8,192	>8,192	
4	72	1	0	0	8	4	9	7	14	12	12	2	1	0	1	0	2	54*
	71†	2	0	0	0	2	0	2	8	15	13	12	11	4	2	1	1	223
	72	3	0	0	1	6	8	10	18	10	10	3	2	1	1	1	1	81
5	66	1	1	0	2	1	9	15	7	10	8	5	0	1	0	1	6	61*
	61†	2	0	0	0	0	1	3	7	15	16	7	6	4	0	0	2	259
	66	3	1	0	0	2	5	11	11	15	12	4	1	3	1	0	0	101
6	53	1	0	0	0	1	5	7	8	13	9	7	2	0	1	0	0	117
	53	2	0	0	0	0	0	2	9	9	12	10	8	1	2	0	0	270
	53	3	0	0	1	2	2	4	9	10	14	5	5	0	1	0	0	142

In Tables I–III titres of >8,192 were taken as 16,384 in calculating the geometric mean.
* Nine Manchester sera with high type 1 titres were excluded in calculating the geometric mean.
† In the case of six subjects there was insufficient serum to titrate type 2 antibody.

had type 1 titres equal to or greater than 1/8,192 and lay well outside the general distribution curve. These high titres were probably due to infection rather than to vaccination, as an epidemic was occurring there at the time, and have been omitted in calculating the geometric mean response. Considering all areas and all batches of vaccine together, the geometric mean response was 1/72, 1/282, and 1/101 for types 1, 2, and 3 respectively.

The results with each of the three batches of vaccine are given in Table II, and it is evident that the antibody responses to all of them were similar.

TABLE III.—*Antibody Titres in Children Under 5 Years of Age and 5 Years and Over After Immunization with Poliomyelitis Vaccine*

Age Group	Vaccine Batch	No. of Children	Geometric Mean Antibody Titre to		
			Type 1	Type 2	Type 3
Under 5 years	4	38	73*	291	91
	5	36			
	6	18			
		92			
5 years and over	4	35	71*	274	110
	5	34			
	6	35			
		104			

From Table III it can be seen that children under 5 years of age responded as well as those aged 5 and over.

Antigenic Activity in Monkeys

The three batches of vaccine were tested for antigenic activity in monkeys by the method in current use in the United States of America (U.S. National Institutes of Health, 1955). A commercial batch of American vaccine, E/2769, kindly supplied by Eli Lilly and Company, was tested at the same time for comparison; this batch was prepared early in 1955, and since then had been stored in the cold without any apparent loss of antigenicity.

For each batch a group of 10 to 12 Rhesus monkeys, weighing between 4 and 8 lb. (1.8 and 3.6 kg.) and with no detectable poliomyelitis antibodies, were given three 1-ml. intramuscular injections of vaccine at intervals of seven days. Sera were obtained seven days after the third injection and titrated for antibody to each of the three types.

TABLE IV.—*Antibody Titres in Rhesus Monkeys After Immunization with Poliomyelitis Vaccine*

Vaccine Batch	No. of Monkeys Injected	Geometric Mean Antibody Titre to:		
		Type 1	Type 2	Type 3
4	12	52	108	32
5	10	67	54	39
6	11	45	47	30
E/2769	10	47	46	24

The results, given in Table IV, show that the antigenic activity of the four batches was very similar in these animals.

Conclusions

It is evident from the results of the study in children that the poliomyelitis vaccine used in this country in 1956 was able to produce an antibody response to all three types of poliomyelitis virus in a high proportion of children. The results compare favourably with those reported from other countries; they are similar to those obtained in America by Salk (1956) after immunization with two doses of vaccine and decidedly better than those reported from Denmark (von Magnus *et al.*, 1955) after intradermal vaccination.

Although both field and laboratory tests indicate that the three different batches of vaccine were similar in antigenic activity, there is so far insufficient information to decide definitely on what response is required in the monkey test in order to ensure that a vaccine will give a substantial response in children. It will be necessary to test, both in the field and in the laboratory, further batches of vaccine

with differing degrees of antigenic activity before a decision on this question is reached. Antigenicity tests in guinea-pigs (Gard *et al.*, 1956) and cotton-rats (Tobin, 1956) are also being investigated in parallel with the field studies.

Investigations are also to be made of the decline of antibody levels in some of the children who took part in the present study to obtain information concerning when a third booster dose should be given.

Summary

A study of the antibody response produced by the three batches of poliomyelitis vaccine used in Britain in the 1956 immunization programme was made in a group of 196 children who had no detectable poliomyelitis antibodies before their course of inoculations.

The children received two doses of vaccine at an interval of three to four weeks, and antibody response to the three types of poliomyelitis virus was determined two weeks after the second dose. No untoward reactions were observed in any of the children as a result of the inoculations.

All except two of the children produced antibodies to all three types, and the average response to each of the three batches of vaccine was similar.

The antigenic activities of the three batches were also shown to be similar when tested in monkeys and of the same order as that of an American vaccine.

The committee is grateful to the parents who consented to have their children vaccinated, and also thanks the Ministry of Health for the supply of poliomyelitis vaccine.

REFERENCES

Enders, J. F., Weller, T. H., and Robbins, F. C. (1952). *Fed. Proc.*, **11**, 467.
Gard, S., Wesslén, T., Fagracus, A., Svedmyr, A., and Olin, G. (1956). *Arch. ges. Virusforsch.*, **6**, 401.
Gear, J. (1956). *S. Afr. med. J.*, **30**, 587.
Sabin, A. B. (1955). *Ann. N.Y. Acad. Sci.*, **61**, 1050.
Salk, J. E. (1956). *Amer. J. publ. Hlth*, **46**, 1.
Tobin, J. O'H. (1956). *Brit. J. exp. Path.*, **37**, 161.
United States National Institutes of Health (1955). "Minimum Requirements for Poliomyelitis Vaccine."
von Magnus, H., von Magnus, P., Petersen, I., Godtfredsen, A., and Rønkjaer, M. (1955). *Dan. med. Bull.*, **2**, 226.

POLIOMYELITIS STUDIES IN AN ISOLATED SCOTTISH COMMUNITY

BY

A. D. MACRAE, M.D., Dip.Bact.

Virus Reference Laboratory, Colindale, London, N.W.9

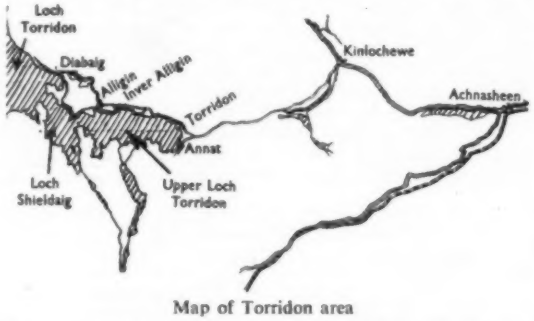
During recent years it has been established that poliomyelitis, spread through human association, is widely disseminated throughout the world. Overcrowding and a low standard of living are associated with the early development of immunity which, once established, appears to persist throughout life. Reinfection may occur to bolster up immunity, but Paul *et al.* (1951), from investigation of an isolated Arctic community lacking opportunities for frequent reinfection, have shown that poliomyelitis antibodies may persist in the blood for many years. The development of these antibodies could be temporally aligned with well-separated outbreaks of the disease in the area.

The natural development of immunity to poliomyelitis is less evident in countries and communities with a higher living standard, and many persons of all ages remain fully or partly susceptible to the disease. The introduction of such persons into an area in which the local population has to a large extent been naturally immunized may result in outbreaks of poliomyelitis involving the new arrivals only. Hammon (1949) has reported this occurrence among recently arrived American children and adults in the Pacific island of Guam. From laboratory studies he found that most native children became infected and developed an immunity below the age of 1, confirming that poliomyelitis was widespread, though mainly inapparent, throughout the island. The circulating viruses showed an apparently increased degree of virulence in the presence of a more susceptible population. He commented on the opportunities for studying the behaviour of poliomyelitis presented by such isolated populations.

Poliomyelitis has been a notifiable disease in this country since 1912, but before 1947 no countrywide epidemics had occurred. In a number of areas there have been no reported cases over prolonged periods, owing either to the existence of a widespread immunity or to lack of exposure to virus. Studies of such a community in the Western Highlands of Scotland in which there had been no evidence of poliomyelitis for many years were planned in 1950. It was hoped by the collection of samples of blood and faeces from members of a representative number of households to determine whether poliomyelitis viruses were currently circulating or had been present in the past.*

Selection of Area

Among the points considered in the selection of a community were the following: (1) the relative remoteness from neighbouring villages and restricted means of approach; (2) the relative compactness of the community itself; too much scatter would add to the problems of collection of specimens; (3) the size and age range of the population; around 400–500 with a reasonable proportion of young people was thought most suitable; (4) the co-operation of the people in supplying information and suitable specimens, and the co-operation of the local medical services; (5) the past incidence of poliomyelitis in the area; (6) any local industries; and (7) the means of transporting specimens to the investigating laboratory.



Map of Torridon area

The area finally selected lay on the north shore of Upper Loch Torridon on the west coast of Ross-shire (see Map). It included the population of Annat, Torridon, Fassag, Corrie, Alligin, and Diabaig. Poliomyelitis had not been diagnosed there within living memory. Approach was by a single road from Kinlochewe, a distance of about 12 miles (19 km.), with the nearest railway station a further 10 miles (16 km.) away at Achnasheen on the route from Dingwall to Kyle of Lochalsh. The local occupations consisted of estate work, crofting, forestry, and fishing. There were few facilities for visitors. Children attended schools in the area up to the age of 11.

*The investigation was planned and the collection of material was organized by Drs. F. O. MacCallum, I. N. Sutherland, and W. C. Cockburn.

The houses in the area were identified on ordnance survey maps and numbered. Lists of the occupants of each house, with details of occupation, age, and school attended if a child, were prepared. In all there were 104 houses with a total population of 294, who had an age range as shown in Table I. Families with one or more children lived in 28 of the houses.

TABLE I.—*Age Range of Torridon Population*

Years:	0–5	6–10	11–15	16–30	31–50	51–91	Total
Males..	13	8	12	21	40	52	146
Females	11	6	6	18	40	67	148
Total	24	14	18	39	80	119	294

Selection of Donors

It was hoped to obtain specimens of blood and faeces from one child in each of the 28 houses, from the youngest child where there were more than one, or failing that from the next youngest, and so on. In addition blood was to be obtained from 30 persons, aged 15 and over, and selected by random sampling from the houses with no children. Specimens of faeces were to be obtained from 12 of these persons. All specimens were to be sent, as quickly as possible after they were obtained by the doctor and nurse, to the nearest bacteriological laboratory. This was 70 miles (112 km.) away, at Inverness. There the serum was to be separated from blood clot and the faeces were to be frozen. The specimens, packed in dry ice, were then to be sent by passenger train to the Virus Reference Laboratory, Colindale.

Collection of Material

An approach, through the doctor and the district nurse, to the persons from whom material for investigation was desired, met with a good response. Specimens of faeces and serum were collected in the spring of 1951, between mid-March and mid-May, and again from the same persons, except for a small number who could not be contacted or who were unwilling, between mid-October and mid-December of the same year. The specimens came from 64 adults and children living in 53 of the 104 houses. There were children under 16 years of age in 25 of the houses. The distribution of the material is shown in Table II. That

TABLE II.—*Distribution of the Material*

Types of Specimen	Age Range in Years						
	0–5	6–10	11–15	16–30	31–50	51–91	Total
Paired sera and stools	4	5	5	2	2	7	25
,, stools only ..	7	—	—	—	—	—	7
,, sera only ..	2	1	—	1	10	9	23
Other combinations	4	1	1	—	—	3	9
Total ..	17 (12)	7 (7)	6 (6)	3 (3)	12 (9)	19 (16)	64 (53)

Figures in parentheses indicate the number of houses from which the specimens came.

samples of serum were not obtained from all children providing faeces was due to difficulty in obtaining blood from some of those under 5 years old. This difficulty had been foreseen, and when it occurred an attempt was made, where possible, to obtain samples of blood from the sibling nearest in age.

Further samples of blood were obtained in the late autumn of 1954 from 19 of the original donors. Of these, four were children up to 5 years old, six were children aged 6 to 10, and three were aged 11 to 15 years; two were adults aged 16 to 30, and four were aged 31 to 50 years. The intention then had been to bleed (1) those less than 20 years old in 1951, and (2) those over 20 years old with either no antibody in their serum against any type of poliomyelitis virus or with antibody to one type only. Early in 1956, samples of blood were obtained from the boy (Table III, No. 6) whose faeces yielded the two strains of poliomyelitis virus in 1951, and from his sister. This family had left Torridon for another locality.

Virus Investigations

The investigations consisted (1) in attempted virus isolation from the samples of faeces and (2) in determining whether poliomyelitis antibodies were present in the serum. The material, collected in 1951, was transported to the laboratory and stored there, frozen at −10° C., until 1954, when it was examined.

Faeces.—A 20% suspension of each sample of faeces in buffered saline solution was centrifuged at 5,000 r.p.m. for at least one hour at 4° C. The supernatant fluid was harvested and tested for the presence of bacteria, being ready for virus investigation when bacteriologically sterile. Twenty-four pools, each composed of equal volumes of three suspensions, were prepared; those, and the remaining single suspension, were investigated in tissue culture and in newborn mice. When a virus was detected in a pool the suspensions composing it were individually studied.

Tissue Culture.—Epithelial cells of rhesus monkey kidney grown from minced tissue fragments or from trypsinized cell suspensions were used in the virus isolation and neutralization tests. Lactalbumin hydrolysate medium (Melnick and Riordan, 1952) or synthetic medium 199 (Morgan *et al.*, 1950) supplemented during the initial period of cell growth with 5% normal horse serum was employed. Tubes were incubated in stationary racks at 37° C. For isolation 0.1-ml. amounts of each pool were inoculated into five tubes, which were then observed microscopically at intervals for cell degeneration. The fluids were harvested when this became apparent or after 10–12 days in its absence. Three tissue culture passes were done before a pool of suspension was regarded as negative. Neutralization tests were based on the method of Ledinko *et al.* (1952), using 100 tissue-culture doses (TCD_{50}) of each virus type against increasing dilutions of serum and leaving each serum–virus mixture at room temperature (about 22° C.) for at least one hour before inoculation into tissue cultures. The virus strains used were Mahoney (type 1), Y–SK (type 2), and Saukett (type 3). The second samples of serum were tested at a single initial dilution of 1 in 2.5 against the three types of poliomyelitis virus and against the newly isolated Coxsackie B3 strain of virus. Where antibody was present against a type of poliomyelitis virus both samples of serum were titrated in increasing half-log dilutions against that virus. In the antibody titrations of the samples of serum obtained in 1954 and 1956 increasing twofold dilutions were used. The serum dilutions given in the tables are the final dilutions after the addition of virus suspension.

Animal Inoculation.—Litters of mice less than 24 hours old were inoculated subcutaneously with 0.02-ml. amounts of each pool suspension or with individual positive suspensions and tissue-culture fluids. Second mouse passages were done when there was evidence of weakness, tremors, or paralysis in the initial passage. Two rhesus monkeys were inoculated intracerebrally (0.5 ml.) and intramuscularly (2 ml. combined with an equal volume of adjuvant —"arlacel A," "bayol F") with tissue-culture fluids of No. 4 or No. 6 strains (Table III). Three rhesus monkeys, each inoculated intracerebrally (0.3 ml.) with faecal suspension from No. 6, were used in the identification of the newly isolated strains of virus from this child.

Results of Virus Isolation

Seven cytopathogenic agents were isolated on first passage in tissue culture from six samples of faeces from young children (Table III). In each instance the faeces was an autumn sample, the earlier specimens from the same children obtained in the spring being negative, as were the other 67 specimens. Five agents were identified as being strains of Coxsackie virus group B, type 3, by neutralization tests using specific antiserum obtained from Dr. Dalldorf. The cell degeneration produced by these strains occurred in a varying proportion of the culture tubes inoculated with each faecal suspension and characteristically

TABLE III.—*Virus Isolations*

No.	Age in Years	House No.	No. in Household	Faeces Sample	Cell Degeneration in 1st T.C. Passage*	Day of Appearance of Degeneration	Virus Identity
1	9/12	54	8†	Autumn, 1951	1/5	5th	Coxsackie B3
2	2 8/12	53	4	,,	1/5	4th	,,
3	8/12	55	3†	,,	4/5	3rd	,,
4‡	5	75	4	,,	1/5	5th	,,
5	8/12	31	6	,,	2/5	10th	,,
6	1 1/12	48	3	,,	5/5	3rd	Poliomyelitis type 2 and type 3

* Number of tubes showing degeneration over number of tubes inoculated.
† All the other persons in these households had Bornholm disease.
‡ Paired sera were available from this child; no serum was obtained in 1951 from the others.

developed slowly in the first passage. In the later passages the degeneration appeared more rapidly and was more complete.

In mice inoculated with pooled faecal suspensions, only one pool which contained the strains of virus No. 1 and No. 2 gave rise to signs of infection. In this instance, weakness, tremors, and varying degrees of paralysis developed after seven days in a litter of six, three of which ultimately recovered, surviving beyond a three-weeks period. In a second passage, using a 20% mouse carcass suspension, the effect was similar, with six mice dying and four recovering. When the five individual faecal suspensions containing Coxsackie virus were inoculated into litters of mice the same slowly developing signs of infection were noted in four instances, but inoculation of further litters with positive tissue-culture fluids yielded more rapid and clear-cut results.

The remaining two agents, isolated from the sixth sample of faeces, were identified as being strains of poliomyelitis virus, type 2 and type 3 respectively. The possibility of this occurrence arose when it was found that serum from the monkey inoculated with tissue-culture fluid of No. 6 strain neutralized types 2 and 3 poliomyelitis virus. The separation of the two strains in monkeys and in tissue culture had already been reported (Macrae and Shepherd, 1955).

Results of Serum Neutralization Tests

The distribution of antibody against the poliomyelitis viruses in the various age groups, based on the serum samples collected in the autumn of 1951, is shown in Table IV. Up to the age of 10, four out of 15 children possessed

TABLE IV.—*Distribution of Neutralizing Antibody to Poliomyelitis Viruses in Serum* of Persons in Different Age Groups in* 1951

Age Range in Years	No. of Persons	Antibody Against Poliomyelitis Virus						
		Type 1	Type 2	Type 3	No Antibody	No. of Types		
						1	2	3
0–5	8 (24)	0	1	0	7	1	0	0
6–10	7 (14)	1	2	0	4	3	0	0
11–15	6 (18)	4	3	1	2	1	2	1
16–30	3 (39)	1	1	1	2	0	0	1
31–50	12 (80)	3	8	4	2	5	5	0
51–91	19 (119)	16	14	16	0	0	11	8
Total	55 (294)	25	29	22	17	10	18	10

* Tested at a final dilution of 1/5.
Figures in parentheses are the total population in each age group.

antibody, in each instance against one virus type. From the age of 11 onwards, a gradual development of antibody, particularly against virus types 1 and 2, was apparent, although the small number of serum samples from the 16–30 year group does not allow a clear picture of events in this age range to be drawn. After the age of 30 exposure to the three types of virus appeared to have been about equal.

Titrations of the paired samples of serum obtained in 1951 were done (1) to determine the individual levels of antibody and (2) to see whether any development of immunity against poliomyelitis virus had occurred during the summer. The results of titration, representing the second samples of serum, are shown in Table V; with one exception (Table VI, No. 2) the titres of the earlier samples of serum showed only minor differences within the limits of experimental error. Of the 19 samples of serum obtained in 1954, 10 were without antibody against any type of poliomyelitis virus; 7 of these were from children who had been less than 10, and one came from a child who had been 15 years old in 1951. The other two sera were from adults who had been 24 and 28 years old.

TABLE V.—*Neutralizing Antibody Levels in the Torridon Sera in* 1951

Poliomyelitis Virus	Range of Serum Dilutions						Total
	<5	−32	−100	−320	−1,000	>1,000	
Type 1	30	10	9	2	3	1	55
,, 2	26	7	8	9	4	1	55
,, 3	33	14	5	2	0	1	55

The remaining nine samples of serum and the two obtained early in 1956 were titrated, together with their preceding 1951 samples, and the results are given in Table VI. These show: (1) that the boy from whom the strains of poliomyelitis virus types 2 and 3 were isolated in 1951 had antibody to these types in 1956; (2) that the sister of this boy had developed antibody to type 2 virus before the collection of her second 1951 sample of serum and had also developed antibody to type 3 virus at a later time; (3) that a boy (No. 5) living in a house close to the home of these children developed antibody to type 2 virus between 1951 and 1954; (4) that a girl (No. 3) with a high antibody level against type 2 virus in 1951 showed a four-fold drop in titre by 1954; and (5) that the other antibody levels showed little variation during the three-year period.

TABLE VI.—*Persistence of Neutralizing Antibody to Poliomyelitis Viruses After an Interval of Three or More Years*

No.	Age (in 1951)	Sex	House No.	Serum and Year	Antibody Titres to Virus		
					Type 1	Type 2	Type 3
1*	1 1/12	M	48	1956	<4	64§	32
2†	5	F	48	1951 A	<4	<4	<4
				1951 B	<4	64	<4
				1956	<4	32	16
3	8	F	77	1951 A	—	1,024	—
				1951 B	<5	1,024	<5
				1954	<2	256	<2
4	9	F	73	1951 A	—	180	—
				1951 B	<5	150	<5
				1954	<2	512	<2
5	10	M	41	1951 A‡	32	<5	<5
				1954	32	<2	<2
6	11	M	47	1951 A	—	<5	—
				1951 B	<5	<5	<5
				1954	<2	256	<2
7	12	F	33	1951 A	256	—	—
				1951 B	512	<5	<5
				1954	256	<2	<2
8	33	M	18	1951 A	—	54	—
				1951 B	<5	46	<5
				1954	<2	54	<2
9	36	F	57	1951 A	—	128	—
				1951 B	<5	90	<5
				1954	<2	56	<2
10	38	M	57	1951 A	—	430	—
				1951 B	<5	256	<5
				1954	<2	256	<2
11	46	F	2	1951 A	2	—	4
				1951 B	2	<5	4
				1954	2	<2	4

* Types 2 and 3 poliomyelitis virus were isolated from this boy in 1951. No sample of serum was obtained then.
† Sister of No. 1.
‡ No sample of serum obtained in autumn, 1951.
§ Reciprocal of serum dilution: calculated by Kaerber's method.

The tests with the samples of serum obtained in the autumn of 1951 showed that 12 contained antibody against the Coxsackie group B type 3 virus. These came from six children less than 10 years old and from six adults ranging in age from 26 to 62. These children, including one (Table III, No. 4) from whom a strain of virus was isolated, and one adult had developed the antibody in the interval between the taking of the first and second specimens, the earlier sera being negative. In three adults the antibody was already present in the spring samples of serum, and in the remaining two adults only the autumn samples of serum were available for testing. The children came from scattered houses throughout the community, but, except for one of pre-school age, attended the same school. A strain of Coxsackie B3 virus had also been isolated from the brother (Table III, No. 1) of one of the children.

In tests with the samples of serum obtained in 1954, 10 persons were found with antibody against the Coxsackie B3 virus. Of these, one was a child (Table III, No. 3) from whom a strain of virus had been isolated in 1951, but from whom no serum had previously been obtained; three were children already positive in 1951; and six were from persons, previously negative, with ages ranging from 9 to 38 years and living in houses scattered throughout the community.

Discussion

Torridon, in which no recorded cases of poliomyelitis have occurred, is a sparsely populated and relatively isolated area. It may, however, be reached from London within 24 hours, without difficulty at most times. As in other Highland communities the population is decreasing, owing to the drift away to the towns in search of work and greater amenities. After the age of 11 the children go elsewhere to complete their education; the young adults stay away because of the lack of work, but some return to settle down in late adult life.

The distribution of antibody to the poliomyelitis viruses appears to bear some relation to this. Up to the age of 11 years, 15 of the 38 children available were investigated serologically; 11 of these had no antibody to any type of virus, and four had antibody against a single type. The three children with type 2 antibody were aged 5, 8, and 9 years, the youngest having developed this during the summer of 1951. One child aged 10 had antibody to type 1 virus. In the 11–15 year group, six of the 18 children available were investigated serologically; four had antibody to type 1 virus, one of these having antibody to all three types, one to type 2 virus, and one to type 3 virus also. As the purpose of the investigation was to sample separate households, the extent of infection in siblings was not estimated. It would appear, however, that infection with type 2 virus had occurred in a number of households during the previous ten years and infection with type 1 virus before that, but there had been no widespread distribution of these viruses.

In the 16–30 year group samples of serum from only three persons were obtained. As one person had antibody to all three types of virus and two were completely negative it is not possible to reach any conclusions. After the age of 30 an increasing proportion of persons had antibody to one or more types of virus, probably acquired elsewhere in a number of instances. The antibody distribution in children differs from that found in Bettws-y-Coed, North Wales (Fallon, 1956), an area from which no cases of poliomyelitis had been notified since 1912. There, antibody against the poliomyelitis viruses, particularly types 1 and 2, was found in the sera of children from the age of 1 onwards, and only 6 out of 57 sera from children under 10 were devoid of antibody.

Although the isolation of two strains of poliomyelitis virus from a boy in the autumn of 1951 showed that these viruses were present in the area, there were no notified cases of poliomyelitis in Torridon during 1951 or subsequently. One adult at Kinlochewe, 12 miles (19 km.) away, was transferred to hospital as a suspected case of non-paralytic poliomyelitis, but no virus was isolated from a sample of faeces obtained while she was in hospital. Serological evidence of recent infection in Torridon was limited to the boy, his sister, and one child from a near-by house who was infected with type 2 virus only. This boy, his sister, and mother had returned to this country from the tropics before the start of the Torridon investigation. No evidence of poliomyelitis infection was found in the material collected in the spring. Whether they had all gone away from Torridon during the summer or whether there had been close contact with relatives or visitors from elsewhere during this time is not known. The age of the children makes it improbable that they had many contacts in the community, hence the limited spread of infection. In addition, there is the possibility, suggested by Dalldorf (1952), of interference with the spread of poliomyelitis virus by Coxsackie B viruses.

Outbreaks of Bornholm disease in 1951, in some instances associated with Coxsackie B3 virus, were reported from Scotland and England (Swain and Mitchell, 1953; Disney *et al.*, 1953). The disease was prevalent in Ross-shire during the summer and autumn, starting on the east coast and spreading westwards. From the Torridon area 13 cases were reported to the medical officer of health (personal communication), including four who were donors of specimens in this investigation. Except for one donor whose serum contained no demonstrable antibody against the Coxsackie B3 virus in either 1951 or 1954, the cases occurred in houses in which evidence of infection with this virus was obtained either by isolation or by antibody development. The laboratory findings support the probability that the clinical illness in Torridon was associated with this type of virus.

The five strains of Coxsackie virus isolated came from young children, three being from near-by houses and two from well-separated houses. One of these was also a combined post-office and shop, the child's father being postmaster, shopkeeper, and, in addition, ferryman and warden of the local youth hostel. The serological findings confirm that infection was widespread, and the combined results are a contrast to the picture with the poliomyelitis viruses. They also serve as a model of what might happen with poliomyelitis, given suitable conditions.

Summary

An investigation of the semi-isolated community of Torridon, in the Highlands of Scotland, for evidence of infection with poliomyelitis is reported. No clinical cases of poliomyelitis have ever been reported from there.

Out of a population of 294 living in 104 houses, samples of faeces, serum, or both were obtained in the spring and again in the autumn of 1951 from 64 persons living in 53 of the houses. Emphasis was placed on obtaining specimens from the youngest child in each family selected. Further samples of serum were obtained in 1954 from 19 of the original donors.

Strains of Coxsackie B3 virus were obtained from five young children living in separate houses, and strains of poliomyelitis virus types 2 and 3 were isolated from one young boy in another house.

The development of antibody against types 2 and 3 poliomyelitis virus during these studies occurred only in the sister of the boy from whom the viruses were isolated, although serum from the boy himself, obtained five years after the virus isolations, did also contain these antibodies. One other child from a near-by house to these children developed antibody against type 2 virus only.

The distribution of antibody in the serum of donors indicated that immunity to poliomyelitis developed

relatively late in life, but that antibody levels, where present, showed little variation over a three-year period.

Serological evidence of widespread recent infections in children and adults with Coxsackie B3 virus corroborated reports of Bornholm disease in the area.

I am indebted to Drs. F. O. MacCallum, I. N. Sutherland, and W. C. Cockburn for advice and encouragement; to Drs. R. D. Martin, D. MacRae (since deceased), J. Landess Horne, Josephine W. Shepherd, H. MacPherson, H. Williams, and the district nurse for their co-operation; and to Dr. G. Dalldorf for the Coxsackie B antiserum.

REFERENCES

Dalldorf, G. (1952). *J. Mt Sinai Hosp.*, **19**, 396.
Disney, M. E., Howard, E. M., Wood, B. S. B., and Findlay, G. M. (1953). *British Medical Journal*, **1**, 1351.
Fallon, R. J. (1956). *Lancet*, **1**, 65.
Hammon, W. M. (1949). *Bact. Rev.*, **13**, 135.
Ledinko, N., Riordan, J. T., and Melnick, J. L. (1952). *Amer. J. Hyg.*, **55**, 323.
Macrae, A. D., and Shepherd, P. A. (1955). *Monthly Bull. Minist. Hlth (Lond.)*, **14**, 55.
Melnick, J. L., and Riordan, J. T. (1952). *Proc. Soc. exp. Biol. (N.Y.)*, **81**, 208.
Morgan, J. F., Morton, H. J., and Parker, R. C. (1950). Ibid., **73**, 1.
Paul, J. R., Riordan, J. T., and Melnick, J. L. (1951). *Amer. J. Hyg.*, **54**, 275.
Swain, R. H. A., and Mitchell, R. G. (1953). *British Medical Journal*, **1**, 1354.

DEATHS FROM POLIOMYELITIS AMONG BRITISH DOCTORS

BY

RICHARD DOLL, M.D., M.R.C.P.
Member of the Statistical Research Unit of the Medical Research Council

AND

A. BRADFORD HILL, C.B.E., F.R.S.
Professor of Medical Statistics, London School of Hygiene and Tropical Medicine; Honorary Director of the Statistical Research Unit of the Medical Research Council

In our study of lung cancer and other causes of death in British doctors in relation to their smoking habits we have observed 34,494 medically qualified men over nearly five years—that is, from November, 1951, to September, 1956, a total of a little more than 165,000 years of exposure.* Over this time and in these 34,494 medical men there have been recorded eight deaths from poliomyelitis (including one certified as acute polioencephalitis). In relation to the size of the population at risk eight is a very small number. Yet it is greatly in excess of the number that would be expected to occur if doctors were dying of poliomyelitis at the same rate as adult males in the general population of Great Britain. Applying to our population of medical men these general population rates in the ten quinquennial groups from age 20 to age 70 (no deaths occurred in the general population over the age of 70) gives an expectation of only 0.83 deaths. It may be that persons in the higher social classes are rather more at risk of contracting poliomyelitis than those in the lower social classes. That would be in accordance with some modern beliefs—though we know of no statistical evidence in this country in its support. But even making them three times as vulnerable we would not expect more than two or three deaths to put against the eight observed. It therefore seems that doctors are specially at risk either of contracting poliomyelitis or, having contracted it, of dying from it.

*In our previous report (Doll and Hill, 1956), the total number of years of exposure observed was referred to as 148,686, but at that time data were available for deaths occurring only until March, 1956.

All those who died were relatively young men. At time of death their ages (last birthday) were 28, 30, 30, 32, 36, 37, 39, and 41. In their work there was no evidence of special risk—for example, in a laboratory. Their last known appointments were hospital casualty officer, hospital medical officer, chest physician, orthopaedic hospital officer, and general practitioner (4). These appointments were widely scattered—London (2), Middlesex, Berkshire, Kent, Staffordshire, Yorkshire, and Lincolnshire. In time, on the other hand, the deaths were curiously concentrated, since five of them occurred in August and September, 1952. The poliomyelitis season in this country is such that one would expect deaths to be specially concentrated in those months, but in each of the 1953 and 1956 seasons there was but one death of a doctor from poliomyelitis and in 1954 and 1955 there was none. The eighth death occurred at the end of the 1951 season (January, 1952). On the other hand, the total number of adult male deaths from poliomyelitis in Great Britain in 1952 (98) was the same as in 1953, not very different from the number in 1955 (105), and only about double that in 1954 (46 deaths) and, it seems likely, 1956.

Summary

The evidence given here suggests that men between the ages of 25 and 45 who are practising medicine in Great Britain are at special risk of dying of poliomyelitis. The actual number of deaths recorded (8) is small in relation to the number of men at risk (nearly 35,000), but at the rates of the general population not more than one death would have been expected to occur.

REFERENCE

Doll, R., and Hill, A. B. (1956). *British Medical Journal*, **2**, 1071.

INVESTIGATION OF NON-ULCER DYSPEPSIA BY GASTRIC BIOPSY

BY

A. WYNN WILLIAMS, M.D., D.C.P. (Lond.)
Senior Lecturer in Pathology, University of Edinburgh

FELICITY EDWARDS, B.M., M.R.C.P.
Research Assistant, West Middlesex Hospital, Isleworth

T. H. C. LEWIS, B.M., B.Ch.
Medical Registrar, West Middlesex Hospital, Isleworth

AND

N. F. COGHILL, M.A., M.B., M.R.C.P.
Physician, West Middlesex Hospital, Isleworth

Large numbers of patients with dyspepsia come to hospital, and often, in spite of appropriate investigations, no cause for it can be found. Although symptoms may abate when a patient learns of negative findings they often persist, and treatment may be difficult and unsatisfactory. A nervous cause of the indigestion may be suspected, but it is difficult to prove. In fact, dyspepsia of unknown aetiology constitutes a notable clinical problem.

Gastritis has long been suggested as a cause of non-ulcer dyspepsia, but in common with others we have found its diagnosis by gastroscopy far from certain (Wirts *et al.*, 1951; Shallenberger *et al.*, 1951; Palmer, 1952; Debray *et al.*, 1953; Selesnick and Kinsella, 1953). The suction biopsy tube introduced by Wood *et al.* (1949) offered a new means of obtaining information

about the gastric mucosa in dyspepsia, and we have used it for this purpose. Details of the modified instrument and method, together with illustrated descriptions of the commoner mucosal lesions, have already been given (Coghill and Wynn Williams, 1955a, 1955b).

By dyspepsia we mean a condition in which abdominal or thoracic symptoms other than dysphagia occur in relation to eating. In this paper non-ulcer dyspepsia (N.U.D.) implies the occurrence of such symptoms when no ulcer can be found in an oesophagus, stomach, or duodenum not previously subjected to surgical intervention. Patients with anaemia, hiatus hernia, oesophageal reflux, carcinoma of the stomach, or hepatic, biliary, pancreatic, intestinal, renal, or cardiac disease were excluded.

Classification of Histopathological Changes in Gastric Mucosa

In our examination of 1,043 biopsy specimens from 467 patients with different disorders we observed changes which were mainly, although not entirely, inflammatory in character. Different classifications of gastritis have been suggested (Schindler, 1947; Magnus, 1952; Palmer, 1954), and these vary in complexity. The following simple classification, which is based partly on that of Motteram (1951), was found to be most useful. It is to be especially noted that in our classification the changes refer to body ("fundic") mucosa.

Chronic Superficial Gastritis.—There is atrophy of the glands in their superficial part and infiltration of the stroma in this region with numerous inflammatory cells, mainly plasma cells and lymphocytes.

Chronic Atrophic Gastritis.—Glandular atrophy is complete or almost so, and there is a variable amount of infiltration of the stroma with inflammatory cells similar in type to those seen in chronic superficial gastritis. Intestinal heterotopia ("metaplasia") commonly occurs, and pyloric gland heterotopia is also of frequent occurrence.

Gastric Atrophy.—The mucosal changes are the same as in chronic atrophic gastritis, but inflammatory cells are inconspicuous.

"Hypertrophic Gastritis."—This unsatisfactory title refers to a group of rare and ill-understood disorders characterized by a marked proliferation of epithelial or stromal cells, or both (Schindler, 1947; Palmer, 1954; Fieber, 1955). In a single example, proved by partial gastrectomy, we were unable to diagnose the condition from biopsy specimens obtained subsequent to operation.

In many patients there were abnormalities in the gastric mucosa which were so slight that classification into any of the groups we have mentioned was not possible. Consequently it was necessary to introduce a fifth group.

Miscellaneous Minor Mucosal Changes.—The glandular pattern is normal, but the surface epithelium or the stroma, or both, are abnormal. The alterations include irregularities in the size, shape, staining properties, and numbers of surface epithelial cells, and oedema and a mild excess of inflammatory cells, usually plasma cells and lymphocytes, in the stroma. The excess of inflammatory cells, in particular, was a common finding.

Material

After excluding patients in whom a cause for the symptoms was found outside the oesophagus, stomach, and duodenum, 775 new subjects with dyspepsia were seen as out-patients in a medical unit over the three years from July 1, 1952, to June 30, 1955—an average annual rate of 258. This figure comprised 25.7% of all new out-patients attending the unit. The corresponding figure for all new general medical out-patients attending the hospital was about 20%.

To assist diagnosis every patient had a barium-meal examination with appropriate investigation of oesophagus and cardia. When there was doubt about the result the examination was repeated and gastroscopy was performed when indicated.

Of the 775 patients with dyspepsia, 459 (59.2%) were men and 316 (40.8%) women. Investigation revealed a likely organic cause for the dyspepsia in 307 (39.6%)—duodenal ulcer, 195 (25%); gastric ulcer, 79 (10%); gastric and duodenal ulcers, 7 (1%); hiatus hernia, 16 (2%); oesophageal reflux, 3; gastric carcinoma, 3; cardiospasm, 2; oesophageal ulcer, 1; volvulus of stomach with eventration of the left diaphragm, 1.

Thus there were 468 patients, forming 60.4% of the whole group, in whom no peptic ulcer could be found, and in whom other causes of dyspepsia were not suggested by the clinical findings or were excluded by appropriate investigations. Of the patients with N.U.D. 251 (53.6%) were men and 217 (46.4%) women.

Gastric biopsy specimens were obtained from 200 of 564 consecutive new out-patients with N.U.D. seen after July 1, 1952. Fourteen had had a peptic ulcer in the past, but none was present at the time of biopsy, although symptoms persisted. Mild overt haemorrhage of the upper alimentary tract had occurred recently in three patients, but in none was notable anaemia present. Attempts at gastric biopsy were made on less than half the patients with N.U.D. There were several reasons for this. Some patients failed to return after their *x*-ray examination, some were too old (Coghill and Wynn Williams, 1955b), and some declined the investigation because they felt better, or for other reasons such as a dislike of swallowing tubes. Some patients with N.U.D. in whom gastric biopsy was performed were grouped under different categories—for example, because of anaemia, as the subject of further studies—and have not been included in this series.

Results

Of the 200 patients with N.U.D. from whom biopsy specimens of the gastric mucosa were obtained, 134 (67%) were men and 66 (33%) women. The mucosa was classified as normal in 90 (45%); 72 (36%) were placed in the miscellaneous group, 9 (4.5%) were classed as chronic superficial gastritis, and 29 (14.5%) were classed as chronic atrophic gastritis. There were no examples of gastric atrophy in the series. In 14 (48%) of the cases of atrophic gastritis the mucosa showed signs of intestinal metaplasia. In 13 patients (6.5% of all cases) multiple specimens demonstrated notably different histology in the same patient, and each case has been classified as showing the more severe of the changes found.

Thus in 110 (55%), rather more than half the patients examined, there was evidence of gastritis. Of these, 72 had only minor changes (miscellaneous group). Major abnormalities (superficial and atrophic gastritis) were found in only 38 patients (19% of the whole N.U.D. group).

Sex.—The distribution between the sexes of patients with normal and different types of abnormal mucosae is shown in Table I and Fig. 1. Fewer women than men were examined, chiefly because fewer presented suffering from N.U.D. and more declined the examination or were unsuitable for

TABLE I.—*Mucosal Findings in 200 Patients with N.U.D. According to Age and Sex*

Age in Years	Males					Females				
	N	M	SG	AG	Total	N	M	SG	AG	Total
18–19	1	2	0	0	3	0	2	0	0	2
20–29	10	8	0	1	19	5	4	0	0	9
30–39	20	12	1	0	33	7	2	1	0	10
40–49	14	10	0	8	32	9	10	1	1	21
50–59	11	6	4	12	33	6	5	1	2	14
60–69	4	5	1	2	12	1	6	0	3	10
70–73	2	0	0	0	2	0	0	0	0	0
Total	62	43	6	23	134	28	29	3	6	66

N = Normal. M = Miscellaneous minor mucosal changes. SG = Chronic superficial gastritis. AG = Chronic atrophic gastritis.

it. Males predominated in about the same proportion in each decade except in the first and the last two. The proportion with minor (miscellaneous group) changes was roughly the same in both sexes. However, fewer women than men showed severe changes.

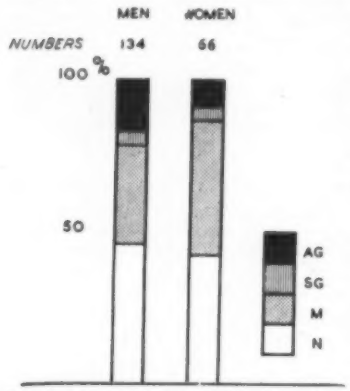


FIG. 1.—Mucosal abnormalities in males and females expressed as a percentage of the total in each group. AG = Chronic atrophic gastritis. SG = Chronic superficial gastritis. M = Miscellaneous minor mucosal changes. N = Normal.

Age.—The mucosal findings are grouped according to the age of the patients in Table I and Fig. 2. Most of the patients were in their fourth, fifth, or sixth decade at the time of examination. Abnormalities were found in the mucosa more often as age advanced. There was a well-marked increase in the incidence of chronic atrophic gastritis with advancing years, except in the group aged 60–73, which contained fewer patients. Mucosal abnormalities are shown by age groups for each sex separately in Fig. 3. Chronic atrophic gastritis was found to be commoner among men in the fifth and sixth decades and in women over 60. The fact that chronic atrophic gastritis is much commoner after the age of 40 is well shown in Fig. 4. Atrophic and superficial gastritis were each present in one out of 55 men under 40, and superficial gastritis in one out of 21 women under 40. Of 79 men over 40, atrophic gastritis was present in 22 (28%), superficial gastritis in 5 (6%), and minor changes in 21 (27%). Of 45 women over 40, atrophic gastritis was found in 6 (13%), superficial gastritis in 2, and minor changes in 21 (47%).

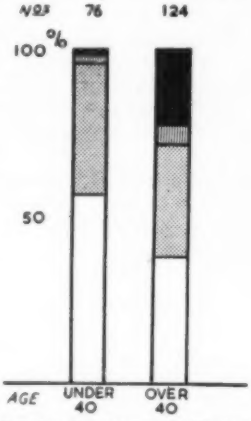


FIG. 4.—Percentage distribution of mucosal changes in patients under and over 40 years of age, irrespective of sex.

Hydrochloric Acid Secretion.—The ability of the stomach to secrete hydrochloric acid was tested in 76.5% of the cases. With the patient fasting, gastric juice was collected through a Ryle tube from the stomach in a resting state and after the subcutaneous injection of 0.5 mg. of histamine. The test was sometimes repeated if no HCl was found. Sometimes 1 mg. of histamine was given. The results are set out in Table II and Fig. 5. They show that patients with minor histological changes in the gastric mucosa (miscellaneous group) failed to secrete HCl under the conditions of the test six times more often than patients with a normal mucous membrane, and patients with severe changes (superficial and atrophic gastritis) failed 15 times more often. Further, when acid was produced it was secreted in smaller quantities the more abnormal the histology. Thus whereas 42.7% of those with a normal mucosa could secrete 21–60 ml. of N/10 HCl, only 16.1% of those with severe histological changes could do so. These findings are close to those of Wood *et al.* (1949) and of Funder and Weiden (1952).

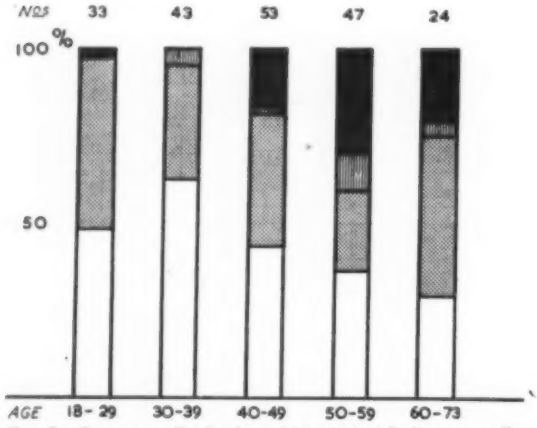


FIG. 2.—Percentage distribution of histological findings according to age, irrespective of sex.

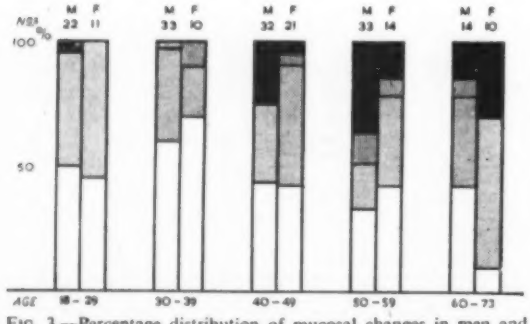


FIG. 3.—Percentage distribution of mucosal changes in men and women according to age.

TABLE II.—*Hydrochloric Acid Secretion in Different Histological Groups*

Histological Group	No. of Patients	No. of Patients Tested	Achlorhydria		Maximum Free HCl in ml. N/10 HCl					
			No.	% of Patients Tested	1–20		21–60		61+	
					No.	%	No.	%	No.	%
N ..	90	68	3	4·4	23	33·8	29	42·7	13	19·1
M ..	72	54	13	24·1	13	24·1	21	38·9	7	13·0
SG ..	9	6	4	66·7	1	12·9	1	16·1	—	—
AG ..	29	25	18	72·0	3		4		—	—

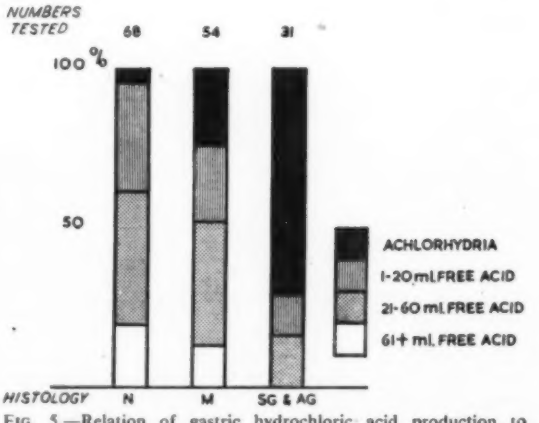


FIG. 5.—Relation of gastric hydrochloric acid production to mucosal changes.

Uniformity of Gastric Mucosa

For assessment of the significance of gastric histological findings it is important to know whether they are diffuse, patchy, or localized. It has been reported that even severe gastritis may be patchy in distribution (Hebbel, 1949a). The method we have used for sampling the mucosa is relatively blind. However, by marking the tube suitably (Wood *et al.*, 1949) it was nearly always possible to obtain samples of body mucosa when required. By taking multiple specimens from the same level after rotating the semi-rigid tube (without withdrawing it), and from different levels, it was possible to obtain a fairly accurate estimate of the homogeneity of the gastric mucosa. Subsequent gastroscopy was sometimes a help in locating biopsy sites.

Joske *et al.* (1955), in a study of 1,000 gastric biopsy specimens obtained with the Australian instrument from a mixed group of patients, reported that in 726 instances when two specimens of mucosa were obtained on the same occasion from different parts of the body of the stomach 73.8% of them were very similar. When specimens were secured on serial occasions from 123 patients the mucosa was substantially the same in 76.4% of cases. Our findings show an even higher percentage of similarity for specimens taken at one time, but less for those taken serially.

We have analysed the appearances of 1,043 biopsy specimens obtained from a mixed group of 467 different patients similar to the Australian series. In cases from which multiple specimens were obtained the mucosal histology has been classed as different if on the separate occasions it fell into different groups as we have defined them. Our findings were as follows :

In 12 patients two specimens were obtained conjoined—that is, the knife had not severed the first specimen completely and the instrument had not rotated properly, so that samples were taken from adjacent areas of mucosa. These specimens all had the same histological appearances.

In 157 patients two or three specimens were obtained at one time from within 1 cm. of the same level, but from different parts of the stomach (mainly anterior and posterior walls). Of these, 144 (91.7%) had the same histological appearances and in 13 (8.3%) they were different.

In 185 patients two to four (occasionally five or six) specimens were obtained at one session from different levels. This figure includes 112 whose specimens came from different levels and 73 whose specimens came from the same and different levels at the same time. Of the former, 101 (90.2%) had the same histological appearances and in 11 (9.8%) they were different. Of the latter, 62 (84.9%) had the same histological appearances and in 11 (15.1%) they were different. Of the specimens from the group of 185 patients as a whole, 163 (88.1%) had the same histological appearances and in 22 (11.9%) they were different.

In 26 patients specimens were obtained at different times. Of these, 11 (42.3%) had the same histological appearances, and in 15 (57.7%) they were different. However, a common reason for securing a biopsy on a further occasion was that the appearances suggested pyloric mucosa. The subsequent specimen was taken from a higher level to be certain of securing body mucosa. This was the case in 6 of the 16 patients with differing histological appearances. Thus in only 38.5% of this group of 26 patients was there an unexplained difference in the biopsy results. Even this is high.

An analysis of the differing histological results is shown in Table III. It was not surprising that in as many as 20 patients normal mucosa was associated with mucosa showing minor changes belonging to our fifth (miscellaneous) category. That atrophic gastritis may sometimes be patchy in distribution is shown by the 17 cases in which it was associated with mucosal specimens presenting only miscellaneous minor changes. Gastric atrophy, when found, was always diffuse.

Although our results indicate that a given biopsy sample was commonly representative of the gastric mucosa, in a minority of cases (10–15%) it was not. As gastritis is not always uniform in distribution, except in pernicious anaemia, increased sampling of the mucosa should produce more discrepant specimens. Analysis of our results on this basis showed that the more samples (but not sites) examined per patient the more likely was atrophic gastritis to be found (Table IV and Fig. 6).

TABLE III.—*Numbers of Patients in Whom Multiple Biopsy Specimens Taken at Same or Different Times Showed Differing Histological Appearances*

	Misc. Minor Lesions	Chronic Superficial Gastritis	Chronic Atrophic Gastritis
Normal	20	2	5
Miscellaneous minor lesions ..		3	17
Chronic superficial gastritis ..			2

Plus two patients with specimens showing normal mucosa and miscellaneous group lesions, with additional specimens showing superficial gastritis in the one case and atrophic gastritis in the other.

TABLE IV.—*Histological Findings when Multiple Biopsy Specimens were Taken, Irrespective of Site or Time, from a Mixed Group of Patients Excluding Cases of Pernicious Anaemia*

No. of Patients	No. of Specimens	Histological Appearances							
		Normal		Misc. Minor Lesions		Chronic Superficial Gastritis		Chronic Atrophic Gastritis	
		No.	%	No.	%	No.	%	No.	%
87	1	42	48·3	27	31·0	7	8·1	11	12·6
236	2	103	43·7	85	36·0	15	6·4	33	14·0
42	3	16	38·1	12	28·6	0	—	14	33·3
55	4	18	32·7	16	29·1	7	12·7	14	25·4
13	5+	1	7·7	5	38·5	0	—	7	53·9

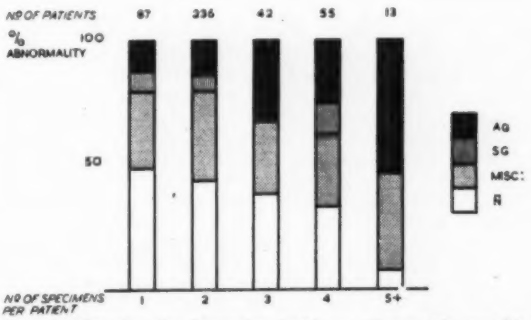


FIG. 6.—Showing the increasing incidence of atrophic gastritis the greater the number of biopsy specimens obtained per patient irrespective of site or time (excluding cases of pernicious anaemia).

Discussion

It is generally acknowledged that dyspepsia is a common complaint, and that in a large proportion of cases no cause can be found. Edwards and Copeman (1943) failed to demonstrate a peptic ulcer in 50% of 436 dyspeptic patients in the Army, while Avery Jones and Pollak (1945) classified 41% of a group of 1,522 patients with gastric symptoms into the category of "non-ulcer dyspepsia." Friedman (1948), reviewing "functional dyspepsia" in the American forces, reported a 10 to 60% incidence in various series of patients, the average incidence for a total of 13,000 patients being 52%. Doll *et al.* (1951), in a survey of 5,951 men and women at work, found 25.4% with N.U.D. compared with 5.6% with peptic ulcer. In our series no cause for the dyspepsia was discovered in 60% of 775 out-patients presenting with this symptom. We have found miscellaneous minor histological changes in the gastric mucosa in 36% of 200 patients with N.U.D. and superficial or atrophic gastritis in 19%. The significance of these findings depends on several factors.

TABLE V.—*Histological Findings in 1,000 Consecutive Gastric Biopsies from a Mixed Group of Patients Classified by Age*

Histological Group	Age Groups													
	0–20		21–30		31–40		41–50		51–60		61–70		71 and Over	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Normal	10	37·0	54	61·4	94	52·8	100	35·6	92	36·1	35	23·5	11	50·0
Miscellaneous minor lesions	14	51·8	29	33·0	59	33·1	111	39·4	72	28·2	36	24·2	2	9·1
Chronic superficial gastritis	0	—	2	2·2	9	5·1	16	5·7	23	9·0	8	5·3	1	4·5
Chronic atrophic gastritis	3	11·2	3	3·4	16	9·0	54	19·3	68	26·7	70	47·0	8	36·4
Total specimens	27		88		178		281		255		149		22	

Two difficulties face the histologist. Firstly, there is the question of whether a few samples of body gastric mucosa are representative of the whole body mucosa. In other words, how diffuse is the lesion in gastritis? Our results, and those of others, show that histological changes in the body mucosa are commonly diffuse, although in a proportion, particularly in the case of atrophic gastritis, they are patchy. They may none the less be scattered diffusely through the body mucosa. We believe that the lesions we found were uniform enough in a sufficient number of patients not to invalidate our general findings. Secondly, there is a lack of complete agreement on the limits of normality in the gastric mucosa at different ages.

Palmer (1952, 1954), using a modified Australian biopsy tube, was the first to obtain material from a sufficient number of "normal" stomachs. While a purist might object that only a small minority of the subjects selected for the investigation were healthy human beings—most were suspected of having amoebic colitis, helminthiasis, or functional colon disturbance—none had clinical, gastroscopic, or radiological evidence of gastric disease. In the vast majority there was a remarkable constancy in the histological appearances: the body ("fundic") gland tubules, with their characteristic chief and parietal cells, were abundant and regular, while only very small numbers of mononuclear cells, including lymphocytes and plasma cells, were present in the stroma. This description is similar to that of normal mucosa generally given in anatomical textbooks, and referring usually to fresh post-mortem tissue, except that Palmer regards even small numbers of plasma cells and lymphocytes in the interglandular stroma as evidence of gastritis. In this respect it is noteworthy that these cells are numerous in the mucosa of normal intestine and yet inflammation is not assumed.

Palmer reported as much normality in the gastric mucosa of 30 subjects over 60 years of age as in the majority of subjects in his investigation who were 40 or under. This high degree of normality in elderly adults is in marked contrast to the figures of most other investigations which are based on study of post-mortem or surgical material. Hebbel (1943) and Guiss and Stewart (1943), for instance, noted an increasing incidence of chronic atrophic gastritis with advancing age in "normal" subjects. The difference is prominent even when allowance is made for the fact that the investigations are not strictly comparable, and no explanation has been given.

We have provisionally accepted Palmer's definition of histological normality for gastric mucosa, and in our classification of lesions seen in our biopsy series we have accepted the usual definitions of chronic atrophic gastritis and gastric atrophy. We have, however, included in the category of "chronic superficial gastritis" only those specimens in which there was atrophy of the superficial part of the glands as well as chronic inflammatory changes. Our fifth group of miscellaneous findings includes relatively minor changes which others (Schindler, 1947; Ylvisaker *et al.*, 1955; Joske *et al.*, 1955) would call superficial gastritis—namely, collections of inflammatory cells in the superficial part of the mucosa without any notable glandular atrophy.

There has been only one account of the gastric mucosa in N.U.D. based on biopsy material with which we can compare our results. Motteram (1951) reported the histological findings in 150 patients with dyspepsia in whom a barium-meal examination had shown no gross abnormality. He describes moderate to severe chronic atrophic gastritis in 33% of patients and "superficial gastritis with minimal atrophy" in a further 30%. From his illustrations, patients in his last group would often have fallen into our fifth group of minor lesions, and we would probably have designated as superficial gastritis a few of the cases in his chronic atrophic gastritis group. Nevertheless, his findings clearly indicate a higher percentage of chronic atrophic gastritis than in our series.

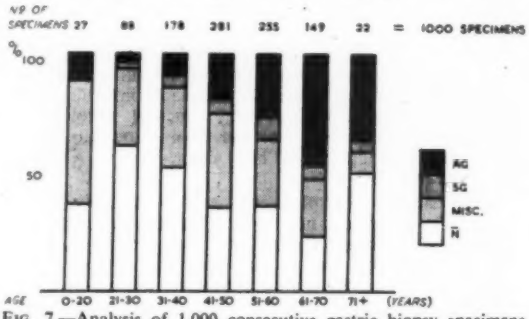


FIG. 7.—Analysis of 1,000 consecutive gastric biopsy specimens according to the age of the patient at the time of biopsy.

Doig and Wood (1952) approached the problem by selecting a group of cases with histological gastritis and examining their clinical aspects; in addition to a few cases with peptic ulcer there were patients with gallstones and other disorders.

On the information at present available we feel it is not reasonable to attach specific clinical significance to gastric

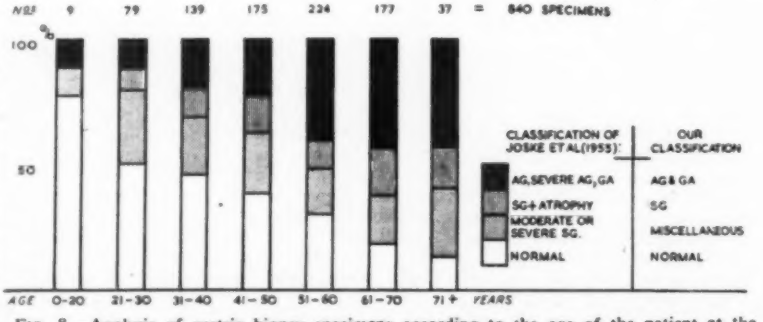


FIG. 8.—Analysis of gastric biopsy specimens according to the age of the patient at the time of biopsy, from Table V of the paper by Joske *et al.* (1955).

mucosal changes. The purpose of this paper is merely to record more information on the condition of the stomach in dyspepsia. Nevertheless there may be clues in the meaning of gastritis if the results of gastric biopsy are examined. In Table V we present, for each 10-year age group, the mucosal changes found in a consecutive series of 1,000 gastric biopsies from 445 different patients suffering from a variety of conditions. These results are set out in Fig. 7. According to the definitions given by Joske *et al.* (1955) it is possible to classify their cases into histological groups closely comparable to our own. In Fig. 8 we present the results from their Table V as a histogram.

Several interesting points emerge from a study of these two series of cases. The incidence of chronic atrophic gastritis was higher in the Australian series; but this is not surprising, because their series included 100 cases of pernicious anaemia and ours only 34. They had more examples of chronic superficial gastritis. In both series the incidence of chronic superficial gastritis and of minor miscellaneous changes was relatively constant, especially during the middle decades (ages 21–70). The constancy with increasing age is even more striking if the numbers in these two last categories are added, particularly in the Australian series. Our general findings in N.U.D. are similar. The incidence of superficial gastritis is much less than that of atrophic gastritis, and there is no progressive increase, in men or women, of either superficial gastritis or miscellaneous minor changes through the decades as there is with atrophic gastritis (Figs. 2 and 3). There are two possible explanations for these results. The histological changes of the fifth (miscellaneous) group and of superficial gastritis might be precursors of chronic atrophic gastritis, there being a similar proportion of patients in each decade with mucosae steadily degenerating and becoming atrophic. Alternatively, these changes might be within normal limits and therefore of no significance. The last suggestion is not supported by the results of tests for gastric hydrochloric acid production. Patients in our fifth group of minor histological lesions had notably inferior power of acid secretion compared with those having a normal mucosa (Table II).

It has been shown by a number of people that in health (Davies and James, 1930; Lerman *et al.*, 1932) and in disease (Vanzant *et al.*, 1932; Hurst, 1932; Ruffin and Dick, 1939) the power to secrete hydrochloric acid declines with advancing years. It has been suggested that this is related to increasing gastric atrophy, and in this connexion it is noteworthy that all workers except Palmer (1954) have found that the incidence of chronic atrophic gastritis increases with age in subjects with or without symptoms. Guiss and Stewart (1948) and Hebbel (1949b) have produced good evidence that the power to secrete hydrochloric acid is usually proportional to the number of parietal cells present. However, the loss of parietal cells is not the only explanation of declining hydrochloric acid production. As Ivy and Grossman (1952) pointed out, it could be due to a reduced stimulating mechanism, although this of itself might lead to atrophy in time.

Summary

Non-ulcer dyspepsia is a common condition for which treatment is often unsatisfactory. Over a period of three years 775 cases of dyspepsia, comprising 26% of new out-patients, were seen: 59% were men and 41% women.

An organic cause for the symptoms was found in 40% (duodenal ulcer 25%; gastric ulcer 10%). Of the 468 patients with N.U.D. remaining, 54% were men and 46% women.

Investigating gastritis as a cause of dyspepsia, we obtained biopsies of the gastric mucosa by means of a modified Australian suction tube in 200 cases of N.U.D.

Categories of mucosal lesions have been defined, and an analysis of the results when multiple specimens were obtained showed agreement between them in 80–90% of cases. There was evidence that the more samples examined the greater was the chance of finding atrophic gastritis.

In the 200 cases of simple uncomplicated N.U.D. the mucosa in 45% was normal. There were mild changes—mainly those of chronic gastritis—in 36% (miscellaneous group), and more severe in 4.5% (superficial gastritis). A diagnosis of chronic atrophic gastritis was made in 14.5%.

There were no notable sex differences in the incidence of gastritis, but the atrophic type became commoner as age advanced. The milder changes occurred about as frequently at all ages, and the significance of these findings is discussed.

Hydrochloric acid production on histamine stimulation fell away as severity of gastritis increased, a usual finding.

Conflicting evidence from the literature makes the interpretation of results difficult. We feel that at present it is best simply to record the findings in this kind of series without drawing conclusions on their relationship to the patients' symptoms.

We thank Dr. A. C. Counsell for his kindness in allowing the histological specimens to be prepared in his laboratory. We are grateful to Professors J. S. Young and G. L. Montgomery for their interest and encouraging help; and to Drs. F. M. Abeles, D. H. Bodger, D. G. Arthur, and M. Spiro for the radiological examinations. We thank Miss Susan Robinson and Mr. D. A. Vinton for help with the charts.

REFERENCES

Coghill, N. F., and Williams, A. Wynn (1955a). *Gastroenterologia (Basel)*, **83**, 60.
—— —— (1955b). *British Medical Journal*, **2**, 1111.
Davies, D. T., and James, T. G. I. (1930). *Quart. J. Med.*, **24**, 1.
Debray, C., Laumonier, R., and Housset, P. (1953). *Sem. Hôp. Paris*, **29**, 2018.
Doig, R. K., and Wood, I. J. (1952). *Med. J. Aust.*, **1**, 593.
Doll, R., Jones, F. A., and Buckatzsch, M. M. (1951). *Spec. Rep. Ser. med. Res. Coun. (Lond.)*, No. 276. H.M.S.O., London.
Edwards, H. C., and Copeman, W. S. C. (1943). *British Medical Journal*, **2**, 640.
Fieber, S. S. (1955). *Gastroenterology*, **28**, 39.
Friedman, M. H. (1948). Ibid., **10**, 586.
Funder, J. F., and Weiden, S. (1952). *Med. J. Aust.*, **1**, 600.
Guiss, L. W., and Stewart, F. W. (1943). *Arch. Surg. (Chicago)*, **46**, 823.
—— —— (1948). Ibid., **57**, 618.
Hebbel, R. (1943). *Amer. J. Path.*, **19**, 43.
—— (1949a). Ibid., **25**, 125.
—— (1949b). *J. nat. Cancer Inst.*, **10**, 505.
Hurst, A. F. (1932). *Quart. J. Med.*, **1**, 157.
Ivy, A. G., and Grossman, M. I. (1952). In *Cowdry's Problems of Ageing*, 3rd ed., edited by A. I. Lansing. Baillière, Tindall and Cox, London.
Jones, F. A., and Pollak, H. (1945). *British Medical Journal*, **1**, 797.
Joske, R. A., Finckh, E. S., and Wood, I. J. (1955). *Quart. J. Med.*, **24**, 269.
Lerman, J., Pierce, F. D., and Brogan, A. J. (1932). *J. clin. Invest.*, **11**, 155.
Magnus, H. A. (1952). In *Modern Trends in Gastro-Enterology*, edited by F. Avery Jones. Butterworth, London.
Motteram, R. (1951). *J. Path. Bact.*, **63**, 389.
Palmer, E. D. (1952). *Gastroenterology*, **21**, 12.
—— (1954). *Medicine (Baltimore)*, **33**, 199
Ruffin, J. M. and Dick, M. (1939). *Ann. Intern. Med.*, **12**, 1940.
Schindler, R. (1947) *Gastritis*. Heinemann London.
Selesnick, S., and Kinsella, E. D. (1953). *New Engl. J. Med.*, **248**, 842.
Shallenberger, P. L., DeWan, C. H., Weed, C. B., and Reganis, J. C. (1951). *Rev. Gastroent.*, **18**, 348.
Vanzant, F. R., Alvarez, W. C., Eusterman, G. B., Dunn, H. L., and Berkson, J. (1932). *Arch. Intern. Med.*, **49**, 345.
Wirts, C. W., Carroll, J. L., and Wald, D. (1951). *Gastroenterology*, **19**, 777.
Wood, I. J., Doig, R. K., Motteram, R., and Hughes, A. (1949). *Lancet*, **1**, 18.
Ylvisaker, R. S., Carey, J. B., sen., Myhre, J., and Carey, J. B., jun. (1955). *Gastroenterology*, **28**, 88.

The Wellcome Foundation Ltd. is to establish a research laboratory in Kenya for the study of diseases of animals in tropical and subtropical areas. At the outset the laboratory will be concerned mainly with problems likely to be amenable to the chemotherapeutic approach. The laboratory will be sited some six miles from Nairobi, within a mile of the Government's veterinary research laboratories at Kabete. Mr. J. K. H. WILDE, formerly deputy director of veterinary research in Nigeria, has been appointed head of the laboratory.

RESPIRATORY VIRUS INFECTIONS IN HOSPITAL AND IN GENERAL PRACTICE, 1954–6

BY

N. R. GRIST, M.B., B.Sc., M.R.C.P.Ed.

AND

R. G. SOMMERVILLE, M.B., Ch.B.

WITH THE TECHNICAL ASSISTANCE OF

H. G. CARSON, F.I.M.L.T.

From the Virus Laboratory of the University Departments of Bacteriology and of Infectious Diseases, Ruchill Hospital, Glasgow

In continuation of previous laboratory studies of respiratory illness, tests for a wider range of virus infections were made in a series of patients admitted to hospital and also in cases of milder illness seen in general practice during the winter seasons of 1954–5 and 1955–6. This paper presents evidence of infections with influenza viruses of types A, B, and C, an agent related to the Sendai strain of newborn pneumonitis virus ("influenza D," Jensen *et al.*, 1955), and the adenovirus group (Rowe *et al.*, 1953).

Methods

The population investigated included 167 patients admitted to pneumonia wards of Ruchill Hospital from July, 1954, to April, 1955, 70 admitted to baby pneumonia wards from November, 1954, to May, 1955, and 98 adults and 44 children under 2 years old admitted to these wards from November, 1955, to April, 1956, inclusive. Tests were also conducted in 105 cases of suspected influenza seen in general practice from December, 1954, to April, 1955, and in 92 similar cases seen in the corresponding five months a year later in the course of schemes for the detection of influenza in the community during vaccine trials of the Medical Research Council (M.R.C. Committee, 1955). A number of additional cases were examined in the course of investigations of outbreaks of respiratory illness. Serum samples were taken during the early and late stages of illness. Garglings for attempted virus isolation were collected with autoclaved skimmed milk or saline with added broth. During the 1955–6 season, respiratory secretions of infants were collected from the nose or throat by sterile cotton-tipped swabs which were subsequently extracted each in a few millilitres of broth for one hour at 37° C.

In general, the cultural and serological techniques were those described in previous papers (Grist, 1955; Grist and Landsman, 1955). For attempted virus isolation, embryonated eggs were inoculated by the amniotic route; at least two passages were made before specimens were regarded as negative.

Sera were examined by small-volume complement-fixation (C.F.) tests and by haemagglutination-inhibition (H.I.) tests. Soluble antigens were used in C.F. tests for influenza A and B; in some experiments soluble antigens prepared from locally isolated strains of virus were used. A fixation period of 90 minutes at 37° C. was employed with adult sera, but with sera from infants the cold overnight method was used. Antigens from several of the following strains of virus were used for H.I. tests: PR8 (type A), A/Scotland/1/53, A/Scotland/26/54, A/Scotland/31/56, Lee (type B), B/England/52, B/Scotland/28/55, and the 1233 strain of type C virus (Taylor, 1949). The Sendai strain of newborn pneumonitis virus was received from the World Influenza Centre of the World Health Organization. Allantoic-fluid antigens of this virus were used in C.F. and H.I. tests; positive and negative control sera for C.F. tests were received from the Virus Laboratory of the School of Public Health, University of Michigan. Type 4 adenovirus (Hilleman and Werner, 1954), together with positive control serum, was received from the Virus Reference Laboratory, Colindale. Adenovirus group antigen was prepared from HeLa cell cultures showing complete degeneration after infection with this virus; the cultures were frozen, thawed, centrifuged at low speed to remove debris, and the supernatant fluid was heated to 56° C. for 30 minutes and stored at 4° C. for use as antigen in the small-volume cold overnight C.F. test. Sera were tested for agglutination of streptococcus M.G. by a standard method similar to that employed at the Virus Reference Laboratory, Colindale. Titres are cited as reciprocals of initial serum dilutions in C.F. and H.I. tests and as reciprocals of final dilutions in streptococcus M.G. agglutination tests.

Results

Influenza

Evidence of influenza infection indicated by fourfold or greater rising titres in serological tests was found during the periods shown in Table I. In common with the rest of Britain, Glasgow experienced an outbreak of influenza B during the winter of 1954–5. A few type A infections detected during April, 1955, heralded the following winter's outbreak of influenza A, which was associated with virus C infections as in the outbreak two years before (Grist, 1955). No influenza infections were found at times other than those shown in Table I. As might be expected, a higher proportion of serologically positive cases was found in the general-practice group than among the miscellany of cases admitted to the pneumonia wards, but very few of the infants showed rising titres of antibody. Thus, during the months when type A or B infections were detected, a specific serological diagnosis of influenza A or B was made in 42% of the general-practice cases, and in 12% of adults, but in only 3% of young children in the hospital series. Two type B strains of influenza virus were isolated during the first winter from garglings collected from children involved in an outbreak of illness in a Glasgow school for the deaf in January, 1955. Dr. A. Harboe, of the World Influenza Centre, reported that these strains closely resembled those sent to the Centre from other countries during the previous two years. During the same period outbreaks of influenza were reported from Ayrshire, and serological evidence of type B infection was found in four out of five cases from which paired sera were sent by Dr. C. D. Rigg, general practitioner.

TABLE I.—*Serological Evidence of Influenza*

Period	Category of Case	No. of Cases Tested	No. with Rising Titres to Indicated Type of Influenza	
Dec., 1954–Mar., 1955	Adult pneumonia	85	12	B
	Baby "	98	2	
	"Influenza"	49	27	
April, 1955	Adult pneumonia	28	1	A
	Baby "	5	0	
	"Influenza"	7	2	
Dec., 1955–Apr., 1956	Adult pneumonia	92	11	A
	Baby "	24	2	
	"Influenza"	92	32	
Jan., 1956–Apr., 1956	Adult pneumonia	89	5	C
	Baby "	19	0	
	"Influenza"	74	1	

During the second winter outbreak eight strains of virus A were isolated: these viruses were identified by Dr. A. Isaacs, of the World Influenza Centre, as Scandinavian subtype A strains similar to those isolated in Glasgow in 1953 and 1954. Two of these viruses were isolated from garglings from adults suffering from clinical influenza and six from throat swabs of infants or young children admitted to the baby pneumonia ward, of whom two aged 1 year showed fourfold or greater increases in antibody to type A virus. Of the other four children, two aged 3 years gave positive

serological reactions in the earliest serum samples and in the second serum of one; no second serum was available from the other child. The remaining two infants were 3 months old: very weak positive reactions were given by the sera of one and no reaction was found with the sera of the other at a dilution of 1/2 in the cold overnight C.F. test with soluble antigens prepared from PR8 or from current (A/Scotland/26/54) strains of virus.

Table II, constructed from the records of adults in both hospital and general practice series from whom paired sera were examined by both C.F. and H.I. tests, shows that these

TABLE II.—*Results of Influenza C.F. and H.I. Tests of Adult Sera*

Season and Type of Influenza	Results of C.F. Tests	Results of H.I. Tests: Fourfold or Greater Rising Titre	Results of H.I. Tests: No Rise of Titre
1954–5 B	Fourfold or greater rising titre ..	28	3
	No rise of titre ..	8	176
1955–6 A	Fourfold or greater rising titre ..	24	15
	No rise of titre ..	8	142

tests were of similar efficiency. Five more cases of influenza B were diagnosed by H.I. than by C.F.; of 31 cases tested by H.I. with two type B antigens, all showed significant increases of antibody titre with current virus antigen (B/Scotland/28/55) and 30 did so with antigen of the Lee strain. Only 62% of type A infections gave diagnostic reactions with both tests, and seven more were detected by C.F. than by H.I.; only antigens from recently isolated strains of virus were used in H.I. tests for influenza A.

A specific serological diagnosis of influenza A or B based on rising titres of antibody was made in 34% of the general-practice cases. When sending blood for examination the practitioners were asked to classify the patients as either "typical" or merely "probable" cases of influenza, basing the distinction on their own clinical judgment. Virus A or B infection was confirmed serologically in 41 (31%) of "probable" and 26 (39%) of "typical" cases.

Newborn Pneumonitis Virus

The detailed results of experience with this agent will be reported separately (Sommerville, to be published). Fourfold or greater rising titres were found by one or both of the C.F. and H.I. tests in three adults admitted to pneumonia wards and also in six of the general-practice cases of suspected influenza (Table III). Of these nine cases of antigenic experience, four were found in the 1954–5 season; the other five occurred during the following winter.

TABLE III.—*Serological Evidence of Infection with Other Respiratory Viruses*

Category	Newborn Pneumonitis Virus: No. Cases Tested	Newborn Pneumonitis Virus: No. with Rising Titres	Adenovirus: No. Cases Tested	Adenovirus: No. with Rising Titres	Streptococcus M.G.: No. Cases Tested	Streptococcus M.G.: No. with Rising Titres
Adult pneumonia	175	3	103	0	199	1
Baby " ..	97	0	32	0	53	0
"Influenza" ..	172	6	70	1	120	0

Adenovirus Infections

C.F. tests with the group antigen were performed with negative results in 103 cases admitted to adult and 32 admitted to baby-pneumonia wards; a rise in titre from <8 to 64 was found in 1 of 70 general-practice cases tested (Table III). One infant pneumonia case admitted in November, 1955, had a C.F. titre of 32 in both serum specimens, suggestive of recent infection. Further evidence of infection with a virus of this group was found during investigation of an outbreak of respiratory illness in a children's residential nursery in Paisley in mid-January, 1956. Eight children aged from 9 months to 4 years became ill on January 14 with fever followed by the development of pneumonia with marked illness and copious nasal and post-nasal discharge. Serological investigations gave no evidence of infection with the viruses of influenza A, B, C, newborn pneumonitis, psittacosis, or Q fever. No influenza virus was isolated from throat swabs by egg inoculation, but in one instance an agent which produced the cytopathogenic effects and gave the C.F. reaction of a member of the adenovirus group was isolated in tissue culture.

The results of C.F. tests on serum from the children (Table IV) show that antibody was detected in the first two sera of the child from whom virus was isolated and that at least

TABLE IV.—*Evidence of Adenovirus Infection in Residential Nursery*

Age of Child (Years)	Virus Isolation from Throat (17/1/56)	C.F. Titres of Sera Tested with Adenovirus Group Antigen: 17/1/56	31/1/56	7/3/56
9/12	–	<5	<5	<8
1	+	40	40	<10
1	–	?20 (a.c.)	<10	<10
2	–	N.T.	N.T.	<8
2	–	?20 (a.c.)	N.T.	10
2	–	N.T.	N.T.	<8
4	–	<5	<10	<8

a.c. = Anticomplementary. N.T. = Not tested.

one other child had some antigenic experience. These findings do not prove that an adenovirus was responsible for the wave of illness in the nursery, although negative C.F. reactions do not exclude the possibility, since Balducci *et al.* (1956) have reported instances of infected children whose sera contained no demonstrable complement-fixing antibodies but nevertheless neutralized the homologous strain of adenovirus isolated. Serological evidence of infection with an adenovirus was also found during investigation of an epidemic of keratoconjunctivitis on Clydeside in the early months of 1956.

Streptococcus M.G. Agglutination Tests

Of 372 cases tested (Table III), none had antibody titres of 40 or greater or showed significantly rising titres except one man aged 24 years who was admitted to the pneumonia ward in March, 1955. Titres of 10, 160, and 320 were found with sera taken on the 4th, 7th, and 15th days of illness respectively; the serum of the 7th day gave a cold agglutination titre of 256. Clinically, the illness was an atypical pneumonia of gradual onset, with right lower lobar consolidation and without leucocytosis.

Discussion

These investigations showed the continuing seasonal activity of influenza viruses in and around Glasgow and again illustrated the tendency of type A and B viruses to become active in alternate years. Virus C infections were again found only during the epidemic of influenza A. Serological evidence of infection with an agent related to the newborn pneumonitis virus, for which the name "influenza D" has been proposed by Jensen *et al.* (1955), was found mainly during the height of the outbreak of influenza A. Significantly high but unchanging titres in the tests for infections with these four viruses were found mainly during and after the periods when cases with fourfold or greater rising titres were detected.

Little evidence of specific viral aetiology for respiratory infections of infants admitted to hospital was found in the present study, but our experience again indicated the greater difficulty in demonstrating serum antibodies in this age group. Not only is the antibody response of infants highly specific for the infecting virus, as compared with the broader response of the antigenically more-experienced adult, but also their antibodies react more weakly than those of older subjects in the C.F. test with influenza antigens (Anderson *et al.*, 1953). In Glasgow, Russell (1956) has found that the period of maximum notifications of pneumonia in children under 1 year old often precedes by some weeks the

seasonal outbreak of influenza which coincides with peak notifications of pneumonia in adults. Similar observations that maximal respiratory illness in young children may not coincide with outbreaks of influenza were reported by Stuart-Harris *et al.* (1949) and by Garrow and Fawcett (1953). The epidemics of acute bronchiolitis in infants in 1951, 1953, and 1955, observed by Heycock and Noble (1956), were all in the pre-Christmas season (Heycock, personal communication), whereas influenza epidemics usually occur later in the winter. It seems likely that influenza viruses are not the main agents responsible for the winter seasonal increase in serious respiratory illness of infants. Nevertheless, influenza infection was detected in several infants in the present study; the clinical aspects of their illnesses will be described in a separate report (Thomas, to be published).

McDonald and Andrews (1955) investigated diagnostic methods in an influenza vaccine trial and concluded that assessments based on purely clinical grounds were apt to underrate while those based on purely serological data were apt to exaggerate the protective power of the vaccine. They recommended that the assessment should be based on the numbers of cases diagnosed as influenza on clinical grounds during the limited period when laboratory tests showed that influenza was present in the community. The general-practice study described above was organized primarily in connexion with vaccine trials. Both hospital and general-practice investigations indicated the activity of influenza viruses in Glasgow during similar periods. A higher proportion of serologically positive cases was found among those seen by the practitioners, and during the second winter influenza A was detected earlier and for a longer period in this group than in hospital. On the other hand, laboratory investigation of patients admitted to hospital with acute respiratory illness is more easily organized, and earlier studies at this laboratory have confirmed that the results of such tests reflect the influenzal experience of the community as judged by other criteria (Grist and Landsman, 1955).

The standard C.F. test with soluble antigen was again found to be a convenient and satisfactory method of diagnosis in adults, though an additional 23% of infections were detected when this test was supplemented by H.I. tests. The difficulty of clinical diagnosis of influenza is illustrated by the similarity in the proportions of confirmed cases in the groups considered "typical" and "probable" influenza by the general practitioners. It should be understood that each practitioner was encouraged to submit specimens from two patients a week whose illnesses might conceivably be influenzal in origin. This was done in order to secure a representative sample of minor febrile and respiratory disease throughout the study, and doubtless resulted in some dilution of influenzal with other cases. Nevertheless, the "typical" group did not contain a significantly higher proportion of cases in which laboratory tests indicated specific infection with influenza virus A or B.

Evidence of infection with members of the adenovirus group has been reported in Britain by Zaiman *et al.* (1955), Andrews *et al.* (1956), and Balducci *et al.* (1956). In the present study these infections were infrequently found except during investigation of an epidemic of keratoconjunctivitis. Rising titres of C.F. antibody were found by Balducci *et al.* (1956) in 3 out of 67 adults with pneumonia or bronchitis in a Sheffield hospital, whereas none of 103 similar cases in the Glasgow hospital study showed a rising titre. The pathogenicity of these viruses for man is incompletely defined, but in this country they have now been associated with the syndromes of febrile catarrh, pharyngo-conjunctival fever (Bell *et al.*, 1955), and epidemic keratoconjunctivitis.

Atypical pneumonia accompanied by the development of cold agglutinins and agglutinins for streptococcus M.G. has continued to be uncommon in Glasgow since these investigations of pneumonia cases began in 1950. Glasgow did not appear to share the epidemic experience of this disease in 1954 reported from East Anglia and London by Wormald *et al.* (1956), Wood (1956), and Garrow (1956).

Summary

Examinations for virus infection were made in 197 cases of suspected influenza seen in general practice and in 265 adults and 114 children admitted to pneumonia wards during the winters of 1954–5 and 1955–6.

Cases of influenza B were detected in all groups during the first winter.

In the second season an outbreak of influenza A occurred; during this period were found several influenza C infections and also instances of antigenic experience with an agent related to the Sendai strain of newborn pneumonitis virus.

Few adenovirus infections were detected except in cases of epidemic keratoconjunctivitis, but a virus of this group was isolated during investigation of an outbreak of respiratory illness in a children's nursery.

One case of atypical pneumonia with agglutinins to streptococcus M.G. was found.

The investigation showed the practicability of detecting the time and type of influenza outbreak by serological tests of cases selected in general practice or admitted to hospital with the diagnosis of pneumonia.

Combined complement-fixation and haemagglutination-inhibition testing revealed more influenza cases than either test alone. The weak immune response of infants made serological diagnosis difficult.

We thank the following practitioners, by whom the general-practice study was carried out: Dr. W. Blair, Dr. W. K. N. Brown, Dr. W. F. Caldwell, Dr. A. Clarke, Dr. S. Dunn, and Dr. T. F. Stewart. For help in other respects we are grateful to our clinical colleagues, particularly Dr. Mary Thomas; to Dr. C. D. Rigg, general practitioner, Dr. Sylvia J. Strachan, of the Public Health Department, Paisley, and Dr. T. S. Wilson, of the Health and Welfare Department, Glasgow, for assistance in investigations of outbreaks of illness; to Dr. C. H. Andrewes, of the World Influenza Centre, Mill Hill, Dr. B. E. Andrews, of the Virus Reference Laboratory, Colindale, and Dr. K. E. Jensen, of the University of Michigan, U.S.A., for supplies of seed virus and control sera; and to our colleagues Lilias G. MacGregor and Helen E. Larminie for assistance with tissue-culture studies.

REFERENCES

Anderson, S. G., Donnelley, M., French, E. L., Kalra, S. L., and White, J. (1953). *Med. J. Aust.*, **2**, 44.
Andrews, B. E., McDonald, J. C., Thorburn, W. B., and Wilson, J. S. (1956). *British Medical Journal*, **1**, 1203.
Balducci, D., Zaiman, E., and Tyrrell, D. A. J. (1956). *Brit. J. exp. Path.*, **37**, 205.
Bell, J. A., Rowe, W. P., Engler, J. I., Parrott, R. H., and Huebner, R. J. (1955). *J. Amer. med. Ass.*, **157**, 1083.
Garrow, D. H. (1956). *British Medical Journal*, **1**, 1105.
—— and Fawcett, J. W. (1953). *Lancet*, **2**, 795.
Grist, N. R. (1955). *British Medical Journal*, **2**, 994.
—— and Landsman, J. B. (1955). *Glasg. med. J.*, **36**, 69.
Heycock, J. B., and Noble, T. C. (1956). *British Medical Journal*, **1**, 438.
Hilleman, M. R., and Werner, J. H. (1954). *Proc. Soc. exp. Biol. (N.Y.)*, **85**, 183.
Jensen, K. E., Minuse, E., and Ackermann, W. W. (1955). *J. Immunol.*, **75**, 71.
McDonald, J. C., and Andrews, B. E. (1955). *British Medical Journal*, **2**, 1232.
M.R.C. Committee (1955). Ibid., **2**, 1139.
Rowe, W. P., Huebner, R. J., Gilmore, L. K., Parrott, R. H., and Ward, T. C. (1953). *Proc. Soc. exp. Biol. (N.Y.)*, **84**, 570.
Russell, S. J. (1957). *Scot. med. J.* In press.
Stuart-Harris, C. H., Laird, J., Tyrrell, D. A. J., Kelsall, M. H., Franks, Z. C., and Pownall, M. (1949). *J. Hyg. (Camb.)*, **47**, 434.
Taylor, R. M. (1949). *Amer. J. publ. Hlth*, **39**, 171.
Wood, P. (1956). *British Medical Journal*, **1**, 711.
Wormald, P. J., Dowsett, L. M., and Walker, J. H. C. (1956). Ibid., **1**, 709.
Zaiman, E., Balducci, D., and Tyrrell, D. A. J. (1955). *Lancet*, **2**, 595.

A film entitled "Hearing Tests in Cerebral Palsied Children" has been made at the Centre for Spastic Children, Chelsea, in co-operation with the audiology unit of the Royal National Throat, Nose, and Ear Hospital. It shows some of the methods and difficulties in testing these children. The film is silent, 16 mm., and 650 ft. (198 m.) in length. A script to accompany the film is available or a lecturer can be supplied. The film is available from the Centre for Spastic Children, 61, Cheyne Walk, Chelsea, London, S.W.3.

INFECTION WITH SENDAI VIRUS IN AN OUTBREAK OF RESPIRATORY ILLNESS

BY

G. B. BRUCE WHITE, M.R.C.S., Dip.Bact.

P. S. GARDNER, M.B., M.R.C.S., Dip.Bact.

Virus Reference Laboratory, Central Public Health Laboratory, Colindale

AND

R. E. HOPE SIMPSON, M.R.C.S.

Director, Epidemiological Unit (Public Health Laboratory Service), Cirencester, Glos.

In February, 1956, an outbreak of respiratory infection, suspected to be due to an influenza virus, occurred in a hospital for the chronic sick and infirm of both sexes (many of limited intelligence). A small section of the establishment also contained rooms for the destitute. In the next month, among 180 patients, 67 cases (25 male, 42 female) were recognized, of which 21 proved fatal. The staff consisted of two part-time doctors, a matron, and 65 full-time and part-time nursing staff, none of whom had an attack of respiratory infection other than minor colds during this period. Specimens of serum were sent to the laboratory for confirmation of influenza virus infection, but no material was collected for virus isolation.

At the same time there were cases of clinical influenza among residents of the town and among nurses at the local cottage hospital. In several of these the diagnosis was confirmed as influenza virus A infection by complement-fixation (C.F.) tests with their sera. When the sera from 41 patients at the "chronic sick" hospital were examined (38 pairs, 3 single convalescent) by C.F. tests and haemagglutination-inhibition (H.A.I.) tests, only 13 of 38 (34%) had rising titres (9 of them fourfold or greater) suggestive of recent infection with influenza virus A. All gave negative results in the streptococcus M.G. agglutination test and in routine C.F. tests with influenza B and C, psittacosis, adenovirus, and *Rickettsia burneti* antigens.

At this time a strain of Sendai virus became available at the Virus Reference Laboratory. This virus, said to be a cause of pneumonitis in infants, was isolated originally in Japan. Complement-fixing and haemagglutinating antigens had been prepared to test sera from infants suffering from acute bronchiolitis with spasm in several outbreaks in the winter of 1955–6. None of these tests had provided evidence of Sendai virus infection, but interesting results were obtained when the same antigens were used to investigate the outbreak in the hospital for chronic sick.

Materials and Methods

The strain of Sendai virus, W.I.C. 12827, was kindly supplied by Dr. Isaacs, of the National Institute for Medical Research, Mill Hill; it had come originally from Dr. Ackermann, School of Public Health, Ann Arbor, Michigan. The strain had had 12 mouse, one rat, two mouse, and 14 egg passages; it had undergone two further egg passages at the Virus Reference Laboratory. The complement-fixing and haemagglutinating antigens consisted of undiluted allantoic fluid derived from 10-day-old fertile hens' eggs inoculated allantoically with 0.1 ml. of a 10^{-4} dilution of stock seed virus. After incubation for three days at 95° F. (35° C.) the eggs were chilled overnight. Fluids containing haemagglutinating virus were pooled, and clarified by centrifugation at low speed.

The strain of mumps virus used in the course of the investigation was that of Enders, E63/2, which had had many egg passages. Haemagglutinating and "virus particle" complement-fixing antigens were prepared by the allantoic inoculation of 8-day-old fertile hens' eggs with 0.1 ml. of a 10^{-2} dilution of stock seed virus. The eggs were incubated at 95° F. (35° C.) for five to six days. They were then chilled and the allantoic fluids were harvested from those eggs containing haemagglutinating virus in high titre. The pooled fluids were spun at 20,000 r.p.m. for one hour after low-speed clarification, and the deposit was resuspended in normal saline in one-tenth the volume of the harvested fluids.

Sodium azide was added as a preservative (in a final concentration of 0.08%) to both mumps and Sendai complement-fixing and haemagglutinating antigens. The optimal dilutions of the antigens for use in the C.F. tests were determined by means of chequerboard titrations with known positive sera.

C.F. tests were carried out in "perspex" plates with cups, using 0.1-ml. quantities of inactivated test serum, complement, and antigen; 2½ M.H.D. of complement were used and fixation of reagents took place for 1½ hours at 37° C. Then 0.2 ml. of 1% sensitized sheep red cells was added and lysis was allowed to occur for a further half-hour at 37° C. Suitable controls were put up with the test, and all results were read next morning after standing at 4° C. overnight.

Sera tested for haemagglutination-inhibiting antibody were first treated for 18 hours at 37° C. with receptor-destroying enzyme (R.D.E.), and after inactivation at 56°–58° C. for one hour were adsorbed in the cold with a suspension of fowl red blood cells which were subsequently removed by low-speed centrifugation.

Haemagglutination-inhibition titrations were carried out in W.H.O. plastic haemagglutination plates (World Health Organization, 1953) employing 0.2-ml. doubling dilutions of the prepared sera in normal saline. Then 0.02 ml. of virus suspension (containing four agglutinating doses of virus) and 0.02 ml. of 7% fowl red blood cells were added to each serum dilution, and the results were read after mixing and standing for 30 minutes at room temperature. Suitable controls were set up with each series of tests.

Results

In consequence of the small number of patients at the hospital providing results suggestive of recent infection with influenza virus A it was decided to retest their sera with a number of other virus antigens. On testing the sera of those elderly patients serologically negative for influenza virus A for their ability to fix complement with Sendai virus preparations, recent infections with this agent were suggested when five patients were found to have rising antibody titres (Table I); two other patients gave positive results at a dilution of 1:16 in both specimens (patients H and F,

TABLE I.—*Results of Complement-fixation and Haemagglutination-inhibition Tests on Patients with Rising Titres to Sendai Virus*

Patient	Complement-fixation Test									Haemagglutination-inhibition Test		
	Influenza A Sol. Antigen			Sendai Antigen			Mumps V Antigen			Sendai Virus		
	1	2	3	1	2	3	1	2	3	1	2	3
Ba ..	4*	4	<8	8	64	<8	8	8	<8	36	288	216
Be ..	—	<8	<8	<8	32	32	—	<8	<8	27	576	216
Sa ..	8	4	<8	<8	16	<8	<8	<8	<8	9	144	144
Hi ..	—	<8	<8	<1	8	<8	—	<1	<8	27	432	—
Ma ..	8	4	8	8	64	16	<8	<8	<8	144	—	>576
Tec. K.	—	<8	<8	<1	16	16	4	4	<8	36	—	432

* Reciprocal of the dilution of sera at which test was positive. In some instances the small quantities of sera available prevented testing at lower dilution than that stated.
— Indicates not tested.

Table II). In addition, the laboratory technician (patient Tec. K) who had collected blood from patient Ba suffered later from a mild respiratory illness and his sera showed rising antibody titres with Sendai virus only. Further examination by C.F. and H.A.I. tests of all remaining sera available from the hospital inmates, and other patients from the town, showed that in all probability a number had been infected recently with Sendai virus, including some who had suffered in addition from an influenza virus A infection. Table II shows those patients with elevated but unchanging antibody titres to Sendai virus, including some with rising antibody titres to influenza virus A.

Examination of further samples of sera (third specimens) collected about three months later confirmed this opinion. The Sendai virus complement-fixing antigen, which had the same constitution as the haemagglutinating antigen—that is, both were undiluted allantoic fluid—gave reproducible results from day to day with sera collected for the investi-

TABLE II.—*Results of Complement-fixation and Haemagglutination-inhibition Tests in Patients Showing Non-rising Titres to Sendai from Hospital and Town*

Patient	Complement-fixation Test									Haemagglutination-inhibition Test		
	Influenza A Sol. Antigen			Sendai Antigen			Mumps V Antigen			Sendai Virus		
	1	2	3	1	2	3	1	2	3	1	2	3
G (H)	<4	16	8	16	16	16	—	<8	<8	108	54	216
K (H)	8	32	16	8	8	<8	—	<8	<8	13·5	13·5	27
H (H)	—	<8	<8	16	16	16	—	<8	<8	432	>576	—
F (H)	<8	<8	<8	16	16	<8	<8	<8	<8	27	108	—
R (H)	<4	<4	16	—	<8	8	—	<8	<8	27	27	54
Ha (T)	<4	<4	—	8	8	—	—	<8	—	144	108	—
L (T)	<2	4	<8	8	8	16	8	8	8	54	108	54
B (T)	<4	8	—	8	8	—	—	<8	—	432	432	—

(H)=hospital; (T)=town.
Sera 1 and 2 were examined in parallel in the C.F. test, but 3 was examined separately owing to shortage of sera. All three were examined in parallel in the H.A.I. test.

gation. In addition, rising titres of haemagglutination-inhibiting antibody always appeared as such on repetition of the tests, but the actual titres obtained tended to vary somewhat when the titrations were repeated on different occasions. This rendered impossible the determination of an H.A.I. baseline value for the detection of patients who had suffered from a previous infection, and from whom only one serum was available or whose paired sera showed unchanging antibody titres. It was not possible to determine exactly the times at which Sendai virus infection started and stopped in the hospital, but the first specimen from Ba, apparently the first case, taken supposedly on the third day of illness, January 20, was already positive at a dilution of 1:8; the sera collected in June (third specimens) from 29 patients providing negative results in February showed no evidence of further rises in antibody titre, with the exception of patient R, whose titre in the third specimen may not be significant because it could not be tested at the same time as the previous specimen. R was also the only patient in whom there was evidence of influenza A virus infection between the first week in March and the third bleeding in June (Table II).

The serum of one of the medical officers, collected in June, did not contain any complement-fixing antibodies at a dilution of 1:8 for influenza virus A antigen, but gave positive results at this dilution with both Sendai virus and mumps "virus particle" antigens. The significance of these findings cannot be interpreted at the moment (see below). Too few sera were obtained from the nursing staff to be able to determine the presence of infection among them.

The sera of 8 of 49 (16%) blood-transfusion donors from the town, collected in June, had Sendai virus complement-fixing titres of 1:8 or higher (five at 1:8 and three at 1:16), suggesting the possibility of infections with this virus in the local population.

While this work was in progress Dr. G. K. Hirst, of New York, visited the laboratory and reminded us that Sendai virus was similar in some respects to Newcastle disease virus. Although Jenson *et al.* (1955) found no serological cross-reactions between Sendai virus and the virus of mumps, the patients' sera were tested for antibody with mumps complement-fixing "virus particle" (V) antigen, which has an antigenic relationship to Newcastle disease virus (Jungherr *et al.*, 1949). It was found that of the six patients in whom rising Sendai virus complement-fixing titres could be demonstrated the sera of five had no complement-fixing antibody at a dilution of 1:8 when tested with mumps V antigen. The sixth patient had an unchanging titre of mumps V antibody of 1:8 in the first two specimens (Table I). The sera of five elderly patients (G, K, H, F, and R, Table II) showing unchanging Sendai virus complement-fixing antibody titres of 1:8 or higher were tested with mumps V antigen, and complement-fixing antibody was found to be absent at a dilution of 1:8.

None of the sera which gave negative results at a dilution of 1:8 in the Sendai virus complement-fixing test (including those with high haemagglutination-inhibiting antibody titres) had mumps V complement-fixing antibody at this dilution. The sera of four patients from the hospital for whom rising Sendai virus haemagglutination-inhibiting and complement-fixing antibody titres could be demonstrated gave negative results when retested with mumps V antigen for haemagglutination-inhibiting antibody.

Among 800 patients with illnesses of all descriptions from whom pairs of sera were received in 1956, five patients were found whose sera showed fourfold or greater increases in complement-fixing titre with Sendai virus and negative (titres <1:8) or non-rising complement-fixing titres with mumps "virus particle" antigen. The sera of these five patients gave negative results when examined routinely for antibodies to influenza viruses A, B, and C, psittacosis, adenovirus, *R. burneti*, lymphocytic choriomeningitis virus, and streptococcus M.G. One of the patients was an adult with an influenza-like illness, and four were children with respiratory infections.

Discussion

In 1953 Kuroya and Ishida described the isolation of a virus from the lungs of a fatal case of pneumonitis in an infant by intranasal inoculation of mice. This virus was isolated subsequently by other Japanese workers from different laboratory stocks of mice, hamsters, and swine. Subsequent investigations (on volunteers) by Japanese workers confirmed that the virus could cause severe pneumonitis in children.

Jensen and his colleagues (1955) found serological evidence of the presence of the virus in the U.S.A. Five cases were identified, but no isolations of virus were made. The results of our preliminary serological surveys of single sera suggest that infection with the virus may not be uncommon in England, but more work is needed to ascertain the significance of these tests. The precise relationship between mumps V and Sendai antigens is being investigated at the moment. It is for this reason that all our suspected cases have been tested with mumps V antigen.

It is of interest that the recent addition to the myxo group of viruses, the "croup agent," isolated by Chanock (1956) in Cincinnati, U.S.A., and by Beale *et al.* (1956) in Toronto, also gives some serological cross-reactions with the mumps virus, but not with Sendai virus.

Summary

In an outbreak of respiratory illness in a hospital for the chronic sick, six patients (five old people and one technician) who developed rising titres of complement-fixing antibody to Sendai virus were observed.

Preliminary surveys show that antibody to Sendai virus is not uncommon in Southern England.

We wish to thank Dr. F. O. MacCallum for his active help and advice in investigating the outbreak and in preparing this paper. We are also indebted to Dr. Preston, Deputy Director, and the staff of the Regional Transfusion Centre, Oxford, for supplying specimens of blood from local donors; and to the laboratory staff at the Memorial Hospital, Cirencester, for the collection and dispatch of specimens.

REFERENCES

Beale, A. J., McLeod, D. L., Stackiw, W., and Rhodes, A. J. (1956). Paper presented at annual meeting, Society of Pediatric Research, Buck Hill Falls, Pa.
Chanock, R. M. (1956). *J. exp. Med.*, **104**, 555.
Jensen, K. E., Minuse, E., and Ackermann, W. W. (1955). *J. Immunol.*, **75**, 71.
Jungherr, E., Luginbuhl, R. E., and Kilham, L. (1949). *Science*, **110**, 333.
Kuroya, M., and Ishida, N. (1953). *Yokohama med. Bull.*, **4**, 217.
World Health Organization (1953). *Tech. Rep. Ser.*, No. 64. Geneva.

SUXAMETHONIUM CHLORIDE ADMINISTRATION AND POST-OPERATIVE MUSCLE PAIN

BY

D. D. B. MORRIS, M.B., F.F.A.R.C.S., D.A.
Senior Anaesthetic Registrar

AND

C. H. DUNN, F.F.A.R.C.S., M.R.C.S., D.A.
Senior Anaesthetist
West Middlesex Hospital, Isleworth, Middlesex

A great deal has been written in praise of suxamethonium chloride ("scoline") as a muscle relaxant in clinical anaesthesia, stressing its freedom from side-effects such as bronchospasm, tachycardia, and fall in blood pressure. In many ways suxamethonium is the ideal relaxant: the rapid onset of complete relaxation followed by the quick return of full muscle power has made it very popular for endotracheal intubation, orthopaedic manipulation, and short abdominal operations. The ideal conditions produced for intubation have undoubtedly reduced the incidence of minor trauma to lips, tongue, and teeth, and of sore throat. One of its principal disadvantages, that of prolonged apnoea, has received so much attention as to obscure other side-effects which produce considerable anaesthetic morbidity.

It is the purpose of this paper to call attention to muscle pain and stiffness, in some cases of great severity, following the use of suxamethonium in patients who are discharged from hospital within 48 hours of operation.

Churchill-Davidson (1954) first drew attention to muscle pains after suxamethonium administration, but this received little attention in the literature; some writers denied that it even occurred. It has been noted that patients kept in bed for two to four days after operation rarely complain of post-operative muscle pains. In the present investigations dental patients discharged the day after operation were chosen. The investigations were designed to show the incidence of post-operative muscle pain, to make an estimate of its severity, and to try to find some simple means of preventing it.

Technique

The patients were premedicated with "omnopon" and scopolamine, or morphine and atropine, in doses appropriate to their age, weight, and physical condition. Induction was with 0.5–0.75 g. of thiopentone 5%, immediately followed by suxamethonium, 50–75 mg., and naso-tracheal intubation. Maintenance was with nitrous oxide, oxygen, and trichlorethylene. On the Nosworthy card was recorded the occupation of the patient, the dose of suxamethonium, and its speed of administration, divided into fast, medium, and slow. The resulting fasciculations were recorded as strong, moderate, and weak, and any particular muscle group strongly affected was noted. The patient's shoulders, chest, abdomen, and legs were bared in order to observe this accurately. The patients were seen the day after operation, when they were discharged, and five days later in the out-patient department, when they attended for routine dental follow-up. The site of any muscle pain or stiffness, its severity, and any spontaneous observations of the patient were recorded.

Control Series

In all, 73 patients were anaesthetized in the way described and followed up. The results were: 25 (35%) complained of pain and/or stiffness on day of discharge; 52 (72%) complained of pain and/or stiffness when seen in out-patient department; and 16 (22%) complained of severe pain and/or stiffness.

The relationship of pain and stiffness to the degree of fasciculations was studied. There seems to be no clear-cut correlation between strong fasciculation and the development of severe pain and stiffness afterwards. Some cases in which the fasciculations were weak developed severe pain and stiffness. One case is of particular interest in this connexion. The only visible fasciculation was an isolated jerk of the rectus muscle, and the patient complained post-operatively that she felt as if she had been "kicked in the belly by a horse." However, when seen in the out-patient department five days later she had experienced generalized aching for three days after discharge. Likewise, there seemed to be no definite relationship between the occupation and the state of physical fitness of the patient. Many athletic patients developed pain, and a blacksmith's striker complained of severe shoulder pain.

Our results are thus in close agreement with Churchill-Davidson's figures—that is, 72% of cases developed pain and stiffness. Our incidence of severe pain was, however, much lower: 22% as compared with 42% approximately. This difference can be explained on the basis that Churchill-Davidson's cases were out-patients, immediately ambulant after operation, whereas ours were in bed for 24 hours after operation. The longer the patient remains in bed after operation the less likely he is to develop suxamethonium pain. One patient, an architect, who had undergone five dental operations before this investigation and developed suxamethonium pain after each one, gave a very interesting account of this phenomenon. He had observed that if he kept still and made no sudden movements he was free of pain; but any activity, such as getting up out of a chair, was painful. Patients quiescent in bed after operation may not notice any discomfort. The figures from the control series show this clearly. Many patients who have no pain on the day of discharge develop it later when they return home. The incidence of pain in patients seen in the out-patient department is twice that on the day of discharge.

Experiments with Modified Technique

The following modifications of anaesthetic technique were made in an attempt to abolish muscle pains:

1. *Chlorpromazine Hydrochloride Experiment.*—Chlorpromazine hydrochloride has a direct paralytic action on skeletal muscle (Burn, 1954). It was hoped that it might modify the painful after-effects of suxamethonium. Chlorpromazine hydrochloride, 50 mg., was given intramuscularly with the premedication. A further 25 mg. was given intravenously on arrival in the theatre, followed by 0.25 g. of thiopentone, 50–75 mg. of suxamethonium, nitrous-oxide, oxygen, and trichlorethylene. Fourteen cases were anaesthetized. The results were: 7 (50%) complained of

pain and/or stiffness on the day of discharge; 8 (56%) complained of pain and/or stiffness when seen in the out-patient department; and none complained of severe pain and/or stiffness. Although this series of cases was very small, administration of chlorpromazine hydrochloride did not seem to influence the incidence of post-operative pain, and the experiment was abandoned on account of the drowsy condition of the patients post-operatively, which in some cases persisted on the morning of discharge.

2. D-*Tubocurarine Chloride Experiment.*—In 65 cases Gray's test dose of D-tubocurarine (5 mg.) was given intravenously before the thiopentone and suxamethonium induction. It is important to observe the effects of these test doses, as a sensitive patient might develop a mixed block on subsequent suxamethonium administration. All of our cases developed ptosis, but none showed hypersensitivity. Visible fasciculations were not abolished in every case: in the few in which they did occur they were weak and transient. The results were: 2 (3%) complained of pain and/or stiffness on the day of discharge; 15 (22%) complained of pain and/or stiffness when seen in out-patient department; and 3 (5%) complained of severe pain and/or stiffness. Churchill-Davidson, in a much smaller series of cases, using 40 mg. of gallamine triethiodide in place of 5 mg. of D-tubocurarine, found that 40% complained of muscle pains, as compared with 22% in this series. There is no doubt that the administration of 5 mg. of D-tubocurarine effectively reduces the incidence and severity of post-operative suxamethonium pain, and is a simple practical measure. The case referred to above in which the patient had experienced suxamethonium pain from five previous anaesthetics underwent a further five dental operations during the investigations. In each of these inductions 5 mg. of D-tubocurarine was given before the thiopentone and suxamethonium induction as described, with complete relief of post-operative muscle pain.

3. D-*Tubocurarine Post-suxamethonium Experiment.*—In order to prove that 5 mg. of D-tubocurarine before suxamethonium is the effective measure in reducing muscle pains, 5 mg. of D-tubocurarine was given after the suxamethonium in 25 cases. No modifications of fasciculations were seen; this was expected, as suxamethonium acts more rapidly than D-tubocurarine. The results, as expected, correlate closely with the control series: 7 (28%) complained of pain and/or stiffness on the day of discharge; 19 (76%) complained of pain and/or stiffness when seen in the out-patient department; and 4 (16%) complained of severe pain and/or stiffness.

4. *Slow Administration Experiment.*—In 27 cases suxamethonium was given at the rate of 10 mg. per 30 seconds until apnoea occurred. When administered in this way fasciculations are greatly reduced in strength. The results were: 12 (44%) complained of pain and/or stiffness on the day of discharge; 20 (74%) complained of pain and/or stiffness when seen in the out-patient department; and 8 (30%) complained of severe pain and/or stiffness. Some of the most severe cases occurred in this group. One female patient was confined to bed on her return home and was so stiff and aching that her relatives had to lift and turn her over in bed. Bourne *et al.* (1952) state that where fasciculations are vigorous post-operative stiffness is more likely. This series shows conclusively that the degree of pain bears no relationship to the strength of visible fasciculations. Six patients who complained of severe pain had no fasciculations at all.

5. *Procaine Experiment.*—Procaine produces neuromuscular block by inhibiting production of acetylcholine at the end-plate. In 25 cases 10–20 ml. of 1% procaine was given intravenously before the thiopentone and suxamethonium induction. The results were: 2 (8%) complained of pain and/or stiffness on the day of discharge; 11 (44%) complained of pain and/or stiffness when seen in the out-patient department; and 3 (12%) complained of severe pain and/or stiffness. Procaine administration does seem to have some influence on suxamethonium muscle pain, but is a clumsy method and not so effective as D-tubocurarine. Procaine administration is time-consuming and suffers from the danger that it may prolong the action of suxamethonium, since they are both destroyed by the same enzyme.

Discussion

It is evident from the results of the investigations that suxamethonium is responsible for considerable post-operative discomfort when a patient is discharged home within 48 hours of its administration. After the patient has been up and walking around for about eight hours, aches and pains start, lasting two or three days; in some cases they are severe and may last for as long as six days. A common description by patients is to say, "I feel as if I had been kicked in the belly by a horse," or "trampled on by a herd of elephants": a tough young man will say, "I feel as if I have been in a brawl." Many patients have called in their own doctors for advice and treatment. The condition usually goes unrecognized, and various diagnostic labels are attached, such as "Fibrositis" or "You must have been in an awkward position on the table."

From the statistical results and from interviews with these patients in the out-patient department it is our firm opinion that suxamethonium should be given up as a relaxant in those cases where the patient is discharged within 48 hours of operation, unless previously modified with D-tubocurarine. Suxamethonium is often the relaxant of choice for a difficult intubation or a manipulation in a very muscular individual, and in these cases the likelihood of suxamethonium pain can be greatly reduced by the use of 5 mg. of D-tubocurarine before the suxamethonium.

Summary

229 cases undergoing dental operation were given suxamethonium chloride ("scoline") for endotracheal intubation, and the incidence of post-operative pain attributable to the drug was investigated.

Slow administration of suxamethonium does not reduce the incidence and severity of post-operative muscle pain.

The only practical and convenient measure to reduce this muscle pain is the administration of 5 mg. of D-tubocurarine or 40 mg. of gallamine triethiodide before the suxamethonium.

We are indebted to Mr. H. Mandiwall, consultant dental surgeon, and Mr. D. A. Brown, senior hospital dental officer, for their co-operation in conducting these investigations.

REFERENCES

Bourne, J. G., Collier, H. O. J., and Somers, G. F. (1952). *Lancet*, **1**, 1225.
Burn, J. H. (1954). *Proc. roy. Soc. Med.*, **47**, 617.
Churchill-Davidson, H. C. (1954). *British Medical Journal*, **1**, 74.

For the first time a section devoted to hospitals will be included in the annual Congress of the Royal Society of Health, which is being held at Folkestone from April 30 to May 3. This section will be presided over by LORD INMAN, Chairman of Charing Cross Hospital. Another innovation will be a forum consisting of a general practitioner, two medical officers, the medical director of a London hospital, and a health visitor, which will discuss questions concerning the health and welfare of the family. During the Congress papers will be read on such subjects as world nutrition, new methods in food preservation, absenteeism in industry, marriage and mental health, slum clearance, food hygiene, fluoridation of public water supplies, and atmospheric pollution. To commemorate the tercentenary of the death of William Harvey, Sir ARTHUR MACNALTY will deliver a special lecture entitled "William Harvey—His Influence on Public Health." Provision has also been made for an overseas forum at which late papers from foreign delegates will be given.

GENERALIZED ARTERIAL CALCIFICATION OF INFANCY

BY

A. C. HUNT, M.D.

Lecturer, Department of Forensic Medicine, the London Hospital Medical College

AND

D. G. LEYS, D.M., F.R.C.P.

Senior Physician, Children's Department, Farnborough Hospital, Kent

In some 40 reported cases of arterial calcification of infancy the proved occurrence in siblings has been described once (Menten and Fetterman, 1948). We report a further example of siblings affected by this disease. It has been described in children of either sex from the age of 1 day to 27 months, usually in the first six months of life. The clinical history and post-mortem appearances in most of the reported examples in early infancy are very similar. The children are born to apparently healthy young parents. The pregnancy and birth are normal, and there is no evidence of abnormal consumption by mother or child of poisons or poisonous dosages of calcifying substances. The infant thrives at first, then, after a few hours, or at the most a few days, of acute symptoms, succumbs from cardiac ischaemia. The symptoms are refusal of feeds, painful rapid respiration, abdominal distension, and heart failure. Occasionally, as in the first of our cases, an electrocardiogram may show the picture typical of coronary occlusion.

Although the clinical manifestations are usually attributable to blockage of the coronary arteries, the calcification affects large and medium-sized arteries throughout the body, and hence we have used the term " generalized arterial calcification."

This is not the only cause of cardiac infarction in infancy, but the syndrome forms a well-defined clinical entity.

Case 1

A male child was born on July 11, 1949. The mother was aged 19 and the father 20; both were in good health with no significant medical history. Both were English and not consanguineous. The child was delivered normally in hospital after an uneventful pregnancy and weighed 7 lb. 13 oz. (3.5 kg.) at birth. He cried vigorously. There was difficulty in establishment at the breast owing to retracted nipples, and the infant was therefore bottle-fed (dried milk). The mother noted no disturbance of any kind until four days before readmission to hospital on August 9, 1949, the infant then being 4 weeks old. The first symptom was refusal of food, and this was followed by vomiting and rapid breathing. The family doctor did not think the child was seriously ill until the day of admission, when respiration became much more difficult.

On arrival at hospital the infant was extremely ill, cyanosed, and pale, with a respiration rate approaching 100, temperature 100° F. (37.8° C.), and heart rate 180–200. The liver was enlarged, but there was no oedema. The clinical diagnosis was of acute infection, possibly accompanied by paroxysmal tachycardia. The cardiogram showed inversion of the T waves and elevation of the ST segment, typical of coronary occlusion. The leucocyte count was 7,000, of which 50% were polymorphonuclears. *X*-ray examination of the chest showed some mottling at the bases. Antibiotics and digoxin were prescribed, but the infant died a few hours later.

Case 2

A third child (a girl) was born at home on October 28, 1955, after a normal pregnancy—a second child (a boy) having been born in 1954. Delivery was spontaneous, and the infant appeared healthy. She was breast-fed and showed no signs of illness until 24 hours before admission to hospital on November 26, then being 4 weeks old. The first symptom was refusal of food, and this was followed by vomiting, the passing of two or three loose stools, and rapid grunting respiration. On admission the infant was cyanosed, pale, and collapsed, with a heart rate of 180, enlargement of liver and spleen, and oedema. There were crepitations over both lungs. The infant died within a few hours.

Post-mortem Examination

Both children were well nourished and externally normal. The most outstanding lesion internally was the calcification of the coronary arteries. The first 2–3 cm. stood out hard, white, and tortuous from a faintly mottled pale-red epicardium. The external iliac, femoral, popliteal, axillary, and brachial arteries were rigidly calcified in almost their whole length, but no other calcification was seen with the naked eye. In Case 2 all of the viscera were radiographed after removal, and, besides the sites already mentioned, calcification was obvious in the mesenteric arteries and in the lingual arteries. The heart of the first child was enlarged (40 g.), predominantly by left ventricular thickening, and the myocardium was firm and light red. In Case 2 the heart was of normal size (23 g.) and the anterior wall of the left ventricle was pale and slightly mottled, the pallor being most pronounced in the inner third. The anterior columnae carniae were flattened. In Case 1 the valves were normal, but in Case 2 there were five minute dark flecks on the contact margin of the posterior mitral cusps. Macroscopically the aorta was normal.

The kidneys were normal to the naked eye. The bones and their epiphysial lines in Case 2 showed no abnormality macroscopically or microscopically. Only the upper pair of parathyroid glands were found in Case 2 despite a prolonged naked-eye and microscopical search. The two found were of normal size and microscopically appeared normal by the criteria of Gilmour (1947). The parathyroid glands were not examined in Case 1.

Microscopical Appearances

Arteries.—The vascular changes in both children were in all major respects similar, although there were minor variations in the degree of involvement at different sites. In addition to the arteries in which calcification was visible, either by naked eye or radiologically, smaller arteries, such as the pericapsular vessels of the thyroid and suprarenals, and arteries in the pancreas and thymus, often showed slight degrees of calcification. The relationship of the calcification to elastic tissue emphasized particularly by Stryker (1946) was very obvious. There was a remarkable predilection for the internal elastic lamina. The minimal lesion was a fine incrustation on each side of the elastic strand, and there were all degrees between this and large irregular masses of calcification distorting the media, with great fibrous intimal proliferation even to the extent of completely obliterating the lumen. In most of the large masses fragments and lengths of elastic tissue could be demonstrated with suitable staining. A common finding was straight bars of calcification along the course of the elastic lamina with loss of the characteristic wavy appearance, and a slight degree of intimal thickening. The pulmonary arteries showed calcification in the elastic strands throughout the media, but no large masses and no intimal changes. A few elastic fibres in the media of the aorta were focally encrusted with calcified material. The affected elastic was at varying levels in the thickness of the aortic media, and sections of all parts of the aorta showed the change. There were no changes in the cerebral vessels. Minimal lesions were present

in the common carotid arteries in Case 1. The elastic laminae remote from the areas of calcification appeared normal.

Heart.—The myocardium of the enlarged heart in Case 1 showed slight patchy interstitial fibrosis, most marked in the subendocardial third and in the papillary muscles, and a few small areas of recent muscle necrosis. In Case 2 there was less fibrosis and more extensive recent infarction, mainly in the anterior wall of the left ventricle. The flecks on the mitral valve consisted of fibrin and red cells, and the valve itself was normal.

Kidneys.—There were amorphous masses of calcification in a few scattered glomeruli in both cases. These masses projected inward from Bowman's capsule and distorted the glomerular tuft. In sections stained with periodic-acid-Schiff the calcification was clearly related to the basement membrane of the capsule, which split to enclose it.

No calcification was found histologically in any other sites, including the gastric mucosa, the pulmonary alveolar walls, and the endocardium.

Examination of the Mother and Surviving Sibling.—This was made clinically, radiologically, and electrocardiographically, and no abnormality was detected. Their serum calcium, blood inorganic phosphorus, and plasma cholesterol were normal.

Discussion

The differential diagnosis clinically between the effects of generalized arterial calcification on the heart and the manifestations of such conditions as glycogen-storage disease, interstitial myocarditis, endocardial fibroelastosis, and aberrant coronary arteries may be difficult. It has been suggested by Cochrane and Bowden (1954) that radiography of the neck may show the arterial calcification.

The aetiology of generalized arterial calcification of infancy is unknown. There is a possibly distinct small group of cases in which primary renal disease may have produced metastatic calcification. Hydronephrosis was found in two children (Bryant and White, 1891; Andersen and Schlesinger, 1942) and congenital renal hypoplasia in one (Cochrane and Bowden, 1954).

A congenital abnormality of the arterial wall and an abnormality of calcium metabolism are the most likely alternatives to account for the remaining cases, and in our opinion the latter is the more probable. The possibility of vitamin D overdosage has been excluded in the majority, although the possibility of hypersensitivity to the vitamin cannot be disproved. The parathyroids have been normal in the few cases in which they have been examined, and no lesions have been found in the bones.

The serum calcium level has been estimated in life in only one example of primary arterial calcification without primary renal disease, and was normal—10.8 mg. per 100 ml. (Traisman *et al.*, 1956). This isolated estimation in a single case cannot be regarded as of great significance.

The predilection of the calcification for elastic tissue has been thought to indicate a primary abnormality of this structure (Stryker, 1946); it has been suggested that the calcification of the arteries in the children with primary renal disease was coincidental. However, calcification round elastic laminae has been described in renal rickets (Shelling and Remsen, 1935), and we have observed a similar picture in material from seven young adults with chronic renal disease and metastatic calcification in the collection of the Bernhard Baron Institute of the London Hospital. In four of these a prominent feature was arterial calcification, most commonly in the form of diffuse calcification of the media, but frequently with a narrow darker staining deposition of calcium against the internal elastic lamina. In all seven cases there were fine incrustations and bars of calcification related to the internal elastic laminae in medium and small arteries closely resembling the lesions of generalized arterial calcification of infancy. It appears that in any state of metastatic calcification the arterial elastic laminae are particularly liable to have calcium deposited round them.

The presence of calcification in the kidneys in many examples of arterial calcification of infancy, either in the glomeruli or in the tubules, suggests that the condition may be part of a generalized metastatic calcification rather than a dystrophic one.

Lightwood (1932) reported widespread arterial calcification in a mentally retarded dwarfed child of 26 months with calcification at sites other than the arteries and kidneys, and with increased density of the bones. The serum calcium was 11 mg. and the blood inorganic phosphorus raised to 6.68 mg. per 100 ml. This case may indicate an aetiological connexion of at least some of the cases of generalized arterial calcification with the severe form of infantile hypercalcaemia described by Fanconi and Girardet (1952), Schlesinger *et al.* (1952), and others.

Furthermore, deposition of calcium in relation to "degenerated" elastica in lingual, meningeal, and renal arteries has been described in a child of 26 months with the severe type of infantile hypercalcaemia (Schlesinger *et al.*, 1956, Case 9).

Until some information becomes available on the biochemical changes in life in generalized arterial calcification of infancy, it is unlikely that any more will be known of its aetiology. It appears from Menten and Fetterman's (1946) cases and ours that the condition may have a familial incidence.

Summary

Two cases are reported of sudden death in infancy from general arterial calcification occurring in a family. Little is known of the aetiology of the condition.

We thank Dr. F. E. Camps for allowing us to use his post-mortem records and material from Case 1.

References

Andersen, D. H., and Schlesinger, E. R. (1942). *Amer. J. Dis. Child.*, **63**, 102.
Bryant, J. H., and White, W. H. (1891). *Guy's Hosp. Rep.*, **55**, 17.
Cochrane, W. A., and Bowden, D. H. (1954). *Pediatrics*, **14**, 222.
Fanconi, G., and Girardet, P. (1952). *Helv. paediat. Acta*, **7**, 314.
Gilmour, J. R. (1947). *Parathyroid Glands and Skeleton in Renal Disease*. London.
Lightwood, R. (1932). *Arch. Dis. Childh.*, **7**, 195.
Menten, M. L., and Fetterman, G. H. (1948). *Amer. J. clin. Path.*, **18**, 805.
Schlesinger, B. E., Butler, N. R., and Black, J. A. (1952). *Helv. pediat Acta*, **7**, 335.
—— —— —— (1956). *British Medical Journal*, **1**, 127.
Shelling, D. H., and Remsen, D. (1935). *Johns Hopk. Hosp. Bull.*, **57**, 158.
Stryker, W. A. (1946). *Amer. J. Path.*, **22**, 1007.
Traisman, H. S., Limperis, N. M., and Traisman, A. S. (1956). *Amer. J. Dis. Child.*, **91**, 34.

"The Flour (Composition) Regulations, 1956, require that all flour, other than wholemeal, shall contain minimum quantities of certain nutrients, including vitamins B_1 and B_2, and iron and chalk. Some of these nutrients are partly removed during milling operations, and the lower extraction flours now being sold require, in many cases, the artificial addition of such substances in order to bring the total amounts in the flour up to the minimal quantities laid down by the Regulations. The nutrients are added by millers in the form of a so-called 'master mix' containing all of them in such amounts as to ensure that if the level of one of them in the flour is of the correct order the remaining ones will also normally be present in the required quantity. From the analytical point of view it will be therefore usually necessary to determine only the amounts of one or two of the nutrients present, and to assume, fairly justifiably, that if these are found to be in the correct proportions the others will be likewise. Four samples of flour were examined during the quarter as a preliminary trial. Three of them were of incorrect composition."—Report of the Birmingham City Analyst.

Medical Memoranda

Primary Dislocation of the Left Shoulder-joint in the Boxing-ring

In the course of a light-heavyweight bout between R.A.F. College, Cranwell, and Cambridge University a cadet was seen suddenly to hold his left elbow with his right glove and to be completely disabled. The round ended almost immediately, and he subsided into the chair in his corner in great pain.

It is often difficult to see some decisive incident in the ring (hence multiple judges as well as a referee), and in this instance I was "stymied" by the referee and saw nothing. As I climbed up to investigate, a colleague in the front row helpfully hazarded the opinion that the man had dislocated his shoulder. On examination this "spot" diagnosis was seen to be perfectly correct, with the classical flattening of the deltoid region and the bulge of the head of the humerus anteromedially and subcoracoid. It was not the moment or the place for bothering the patient about the detailed mechanism whereby this extraordinary happening had come about. I was puzzled because, in boxing, the *left* upper limb is mainly concerned with the safe piston-like movements of leading and guarding, in which no considerable abduction is involved. It would have been much more explicable in the case of the *right* shoulder-joint, where wide abduction occurs in swinging a right hook to the jaw. Moreover, this was not a recurrence. The man had never dislocated his shoulder before, and so lacked the notorious tendency to recurrence. I wrote to the patient and to the S.M.O. at Cranwell R.A.F. College, asking if they could throw any further light on the dislocation, and I also questioned his opponent, our own light-heavyweight.

The cadet replied: "It was very difficult, as I didn't actually feel it happen at all. The only sensation was of not being able to raise my left arm, and then I thought it must be cramp. I didn't realize the shoulder was dislocated until I returned to my corner at the end of the round." He continued: "I have asked independently all the members of the team who were watching, and . . . it appears that . . . I attempted a left hook, and while my arm was fully extended (my punch having missed) he hit my arm with a right-hand punch."

The opponent confirmed this missed hook, and stated that he himself had a very sore right ulna from the violent impact of his own upthrusting *forearm* blow in warding off the said hook.

COMMENT

It would appear that the patient's left shoulder-joint was relaxed, with flaccid muscular control, at the moment when his arm was widely abducted and the blow missed. Simultaneously the Cambridge man violently forced the arm much further upwards, bruising his own forearm, and levering the head of the humerus downwards out of its shallow socket and through the capsule of the joint.

It is hardly necessary to recall that the stability of the shoulder-joint (with its shallow glenoid cavity providing little or nothing to hold the head of the humerus) depends on the tension of the tendons passing under, over, or through the joint and the elastic compressing pull of the muscles acting upon it. The dislocation happened at the moment of misfire, when all these structures were relaxed.

I have never seen this accident in the boxing-ring before, during thirty years at the Cambridge ringside, nor have I heard of it elsewhere. It would seem that primary dislocation of the shoulder-joint in the boxing-ring is an extremely rare accident, comparable in rarity with dislocation of the hip-joint at "rugger" (Woods, 1949).

R. SALISBURY WOODS, M.D., F.R.C.S.,
Honorary Medical Officer,
Cambridge University Boxing Club.

REFERENCE

Woods, R. S. (1949). *British Medical Journal*, 1, 272.

Agranulocytosis During Treatment with an Amoebicide

A case of agranulocytosis developing in the course of treatment of amoebiasis with "camoform" is described. It appears to be the first time in which such a haematological change has been noted during camoform medication.

CASE REPORT

In November, 1955, in Calcutta, an Englishwoman aged 27 complained of abdominal discomfort and dyspepsia. She had never been ill before. She had one child aged 15 months. On clinical examination no abnormality except moderate obesity was found. A stool examination revealed mucus and a few precystic and cystic forms of *Entamoeba histolytica*, no cellular exudate, and no ova. A blood count showed: erythrocytes, 4,510,000; Hb, 12.6 g./100 ml.; leucocytes, 5,200 (polymorphs 70%, lymphocytes 27%, monocytes 0%, eosinophils 3%). Her urine contained no abnormality. Camoform, one tablet (250 mg.) four times a day, was prescribed. On the fifth day of treatment her temperature was 101° F. (38.3° C.), and she had mild photophobia and mild pains in the limbs and neck. On the following day the fever had abated to 100° F. (37.3° C.), and she had very little photophobia or pain. She did not complain of sore throat and no pharyngeal abnormality was found on examination. On that day her leucocytes numbered 2,300 (polymorphs 71%, lymphocytes 24%, monocytes 3%, eosinophils 2%).

Camoform treatment was stopped after a total of 16 tablets (4 g.) had been taken. On the sixth day the leucocytes had further decreased to 2,000 (polymorphs 56%, lymphocytes 40%, monocytes 2%, eosinophils 2%). On the seventh day the lowest readings were obtained: leucocytes, 1,200 (polymorphs 56%, lymphocytes 40%, monocytes 1%, eosinophils 3%).

The patient was admitted to hospital on the sixth day and treatment instituted. Penicillin was given eight-hourly in doses of 400,000 units intramuscularly (crystalline penicillin G procaine 300,000 units, with buffered crystalline penicillin G potassium 100,000 units) for five days. "Pentnucleotide," 10 ml. daily, was given intramuscularly for the first three days. Cortisone acetate, 150 mg. daily in divided doses, was also given for four days. Afterwards the dose of cortisone acetate was gradually reduced. On the third day of this treatment the leucocytes rose to 1,800 (polymorphs 68%, lymphocytes 30%, monocytes 1%, eosinophils 1%), and there was a consistent improvement onwards. Five days later a blood count showed: erythrocytes, 4,250,000; Hb, 12 g./100 ml.; leucocytes, 5,100 (polymorphs 54%, lymphocytes 34%, monocytes 2%, eosinophils 10%). Four months later the patient was still well.

COMMENT

In a series of 20 cases Barrios (1954) reported no haematological toxic signs, though three of his patients developed anorexia, transient albuminuria, or headache. Abdominal pain, headache, nausea, dizziness or vertigo, sweating, or palpitation were observed in East Africa by De Mello and De Mello (1955), but they, too, do not report any blood dyscrasia. In Honduras, Hoekenga and Batterton (1954) gave camoform, 1.5 g. daily for seven days, to a series of 18 patients with amoebiasis, with no ill effect.

We have used camoform in a large number of cases of amoebiasis, but this case of agranulocytosis is the only one in which a serious toxic reaction followed the use of the drug.

Acknowledgment is made to Professor B. P. Tribedi for his help in performing laboratory investigations; to Messrs. Parke, Davis and Co. Ltd.; and to the Librarian of the Royal Society of Medicine for aid in surveying relevant literature.

H. F. HAHNDEL, M.D.
L. PREGER, M.B., D.Obst.R.C.O.G.

REFERENCES

Barrios, H. (1954). *Gastroenterology*, 27, 81.
De Mello, J. P., and De Mello, Ruby, N. (1955). *E. Afr. med. J.*, 32, 47.
Hoekenga, M. T. and Batterton, D. L. (1954). *Amer. J. trop. Med. Hyg.*, 3, 849.

Reviews

ANATOMY AND PHYSIOLOGY OF GALEN

Galen on Anatomical Procedures. Translation of the surviving books with introduction and notes by Charles Singer. (Pp. 289+xxxvi; illustrated. 37s. 6d.) London, New York, Toronto: Oxford University Press for Wellcome Historical Medical Museum. 1956.

This translation by Dr. Charles Singer of Galen's *De Anatomicis Administrationibus* is a very useful addition to the publications sponsored by the Wellcome Historical Medical Museum. In it we have what is essentially Galen's "Little Cunningham," for the book is a translation of a series of lecture-demonstrations on anatomy and physiology given at Rome in the year A.D. 177. The greater part of it comprises a dissecting manual, not of the human but of the monkey or ape, as Galen himself freely acknowledges. This is not to say that Galen did not have access to human material in his studies, for he indicates quite clearly that he had worked on the human skeleton—and there are other hints that he knew something of the soft parts. Such knowledge must have been obtained as occasion offered, and so he officially advises his students to dissect either the Barbary ape or the rhesus monkey, which seemed to him most like the human in structure. Professor Singer has very carefully assessed the problem to which of these two creatures the majority of Galen's dissecting instructions refer—and he believes that the animals used were rhesus monkeys. Accordingly the text has been illustrated by relevant figures from the excellent monograph by Hartman and Straus (*The Anatomy of the Rhesus Monkey*, 1953), which do much to assist the reader.

One of Galen's difficulties was that of nomenclature, especially with regard to the muscles. There were few "official" names for many structures when he wrote, so his descriptions may seem rather verbose and the structures difficult for us to identify, the more so since his observations do not refer to the human; but Singer has interpolated the modern names in the text, and in a series of notes has shown the essential differences between monkey and man for those who know little of comparative anatomy. Much of the value of the translation stems from this scholarly work. Galen, however, was not only an investigator of the dead body; he constantly refers to observations on the living human where wounds or disease are present; and, again, he practised operations on animals in attempts to understand the mode of the circulation and respiration. In this he showed the essential linkage between anatomy and physiology as two subdivisions of one science.

This book should be in all medical libraries; it would be worth while for postgraduate and undergraduate students to study it at their leisure, and to learn from it that the sciences of anatomy and physiology are not of such recent development as they may think, and reflect on the heritage to which they are successors.

J. S. BAXTER.

THE HEART AND ITS DISORDERS

Cardiology. By William Evans, M.D., D.Sc., F.R.C.P. Second edition. (Pp. 574+x; illustrated. 92s. 6d.) London: Butterworth and Co. Ltd. 1956.

Diseases of the Heart and Circulation. By Paul Wood, O.B.E., M.D., F.R.C.P. Second edition. (Pp. 1,005+xxxviii; illustrated. £5 5s.) London: Eyre and Spottiswoode Ltd. 1956.

That new editions of two books emanating from the National Heart Hospital should appear in the same month is a testimony to the authority, ability, and industry of their distinguished authors. Both dedicated to Sir John Parkinson, the doyen of British cardiologists, both nurtured and maturing in the same stable, yet were there ever such dissimilar but attractive twins?

William Evans claims to write for the student requiring a concise statement of modern views. Dogmatic, personal, steeped in the clinical outlook, more than a trifle prejudiced at times, but always vastly stimulating, his book sets out his views with a deep conviction and a burning fervour. This is the gospel according to Evans. Not the least praiseworthy of his many attributes is the author's nonconformist attitude, which should serve to arouse the critical powers of the more experienced postgraduate. A most commendable, original, but unorthodox book by a great clinical teacher.

Paul Wood writes primarily for the graduate, and modestly claims that his volume is not intended for the consultant cardiologist. In point of fact few physicians—cardiologists or not—can afford to dispense with this book, which provides a comprehensive survey, unique in its scope and outlook. Similarly, surgeons, now entering this field in increasing numbers and not averse to advice, will find the help and guidance they require. The author brings to the bedside a vital dynamic approach, based primarily on the application to man of the physiological techniques which have revolutionized our concepts of circulatory mechanisms over the past 20 or 30 years. Facts and figures, opinions and hypotheses, are criticized and analysed with a masterly assurance. This is the naked truth according to Wood. In breadth of outlook, in the blending of clinical experience with the results of technical procedures, and in their welding together, a sound critical and constructive judgment is revealed. There is no better presentation of the advance in our understanding of circulatory disease than this masterly survey from the pen of Paul Wood—an outstanding achievement which brings great credit to British cardiology.

A. RAE GILCHRIST.

NON-VENEREAL GENITAL DISEASES

The Nonvenereal Diseases of the Genitals: Etiology, Differential Diagnosis and Therapy. By Fritz T. Callomon, M.D., and John F. Wilson, B.S., M.D., M.S. (Pp. 382+xiii; illustrated. 95s.) Springfield, Illinois: Charles C. Thomas. Oxford: Blackwell Scientific Publications. 1956.

This is a book by two dermatologists who have undertaken to give a detailed comprehensive description of all the nonvenereal affections of the genitalia, whether localized to that area or forming part of more generalized conditions. They have covered a wide field, and some of their descriptions, especially of dermatological conditions, are excellent. Disproportionate attention is, however, paid to the rare and unusual, of which the description is in some cases unduly discursive, with the result that the book is far longer than the subject-matter really requires. Its chief value will be as a book of reference, and as such it is particularly commended to venereologists, for all kinds of lesions which have nothing to do with venereal infection are apt to present in the clinic for venereal diseases. There is also much of interest to the dermatologist, the gynaecologist, and the urologist.

The sections on treatment are valuable and in most instances well up to date. In general the English is lucid enough, but it is sometimes quaintly phrased, almost as though it had been translated too literally from a foreign language. There are a number of typographical errors which careful proof-reading might have corrected, and the references, though large in number, are not always accurate and seem to show a bias, perhaps determined by a nostalgic devotion to out-of-date German literature. Many of the illustrations are excellent, but others are not so. Those readers who feel able to afford the considerable cost of a personal copy will be rewarded with a book that is well printed and produced on excellent paper, and subject-matter which contains much of interest. It could, however, have been much better done, for when the authors deal with some subjects outside the immediate scope of dermatology, such as non-gonococcal urethritis, the standards of achievement are very indifferent.

AMBROSE J. KING.

CLINICAL ORTHOPAEDICS

Clinical Orthopaedics—No. 7. Edited by Anthony F. DePalma, with the assistance of the associate editors, the board of advisory editors, and the board of corresponding editors. (Pp. 354+x; illustrated. 60s., or 48s. to subscribers.) Philadelphia and Montreal: J. B. Lippincott Company. London: Pitman Medical Publishing Company, Ltd. 1956.

The subject-matter in No. 7 of this series is bone tumours and injuries resulting from motor accidents. There are, in addition, an appreciation of Bloodgood's work and a few chapters on general orthopaedic subjects, including central fracture of the acetabulum. There is not a textbook description of all bone tumours, but those of interest are chosen, either because there has been particular study of such growths as chordoma and osteoid osteoma, or because the advance of knowledge is slow, as in the case of the sarcomata. Copeland writes on tumours of cartilaginous origin, and Jaffe and others on osteogenic sarcoma. It is reported that solitary and multiple osteochondromata and chondromatosis of joints may acquire malignant changes. The history of several types of sarcomata is well described. Biopsy is advocated to provide accurate diagnosis rather on the assumption that the manipulation during examination is a more potent factor in dissemination than the operation. The round-cell sarcomata are separated into types, and it is stated that correct diagnosis is essential, as the cell type of the neoplasm determines the nature of the appropriate treatment. There are reports on 107 fibrosarcomata and 32 aneurysmal bone cysts from the Mayo Clinic and of a chondroblastoma of the talus from Texas.

Ample material was available for the study of several aspects of motor injury, such as the time intervals between injury and death, the crush syndrome, and the injuries of young and old. Readers will be interested in chapters on head, chest, and abdominal injuries. The information on residual disabilities will be accepted, but the opinion expressed on the withholding of a driving licence on medical grounds is unlikely to meet with approval in this country.

These volumes continue to contain high-grade information, and the illustrations, especially the reproductions of radiographs, are excellent.

ST. J. D. BUXTON.

DISEASES OF THE SKIN

An Introduction to Dermatology. By G. H. Percival, M.D., Ph.D., F.R.C.P., D.P.H. Twelfth edition. (Pp. 374+vii; illustrated. 45s.) Edinburgh and London: E. and S. Livingstone Ltd. 1956.

The Edinburgh school of dermatology is well established, having been founded by William Allan Jamieson and carried on by his successor, the late Sir Norman Walker. Both wrote textbooks on diseases of the skin and made the Edinburgh teaching more generally available. It is now over half a century since Sir Norman Walker's first edition of *An Introduction to Dermatology* was published. Professor Percival, who holds the only chair of dermatology in the British Isles, collaborated with him in the production of the tenth edition, and nine years ago was solely responsible for the eleventh. Since then additional antibiotics, the discovery of A.C.T.H. and cortisone, and the appearance of other new therapeutic agents have made a new edition essential and welcome.

In the twelfth edition the arrangement of the contents is unchanged, the chapter headings being precisely the same as before. There are 25 more pages and six more figures. In a book of this kind the illustrations are of great importance. Almost without exception these are excellent, and it is remarkable that 188 of them are in full colour. The book is well balanced in its consideration of both common and rare conditions. It is, moreover, essentially practical in its approach, and once more can be warmly recommended to medical students and general practitioners, for whom it is written.

S. T. ANNING.

PHYSICAL AIDS IN PLASTIC SURGERY

Physical Methods in Plastic Surgery. By Joseph P. Reidy, M.A., M.B., B.Ch.(Cantab.), F.R.C.S. General editor of physical medicine series: William Beaumont, M.R.C.S., L.R.C.P. (Pp. 69+xiii; illustrated. 12s. 6d.) London: The Actinic Press. 1956.

Although the physiotherapist, the occupational therapist, the remedial gymnast, and the rehabilitation expert may all have their parts to play in the team which is concerned with the treatment of patients requiring reconstructive or plastic procedures, it is often extremely difficult to define their existing activities. These, in any case, will vary from patient to patient and from condition to condition. Instead of producing a dry catalogue of the possible remedial procedures, the author has written an informative little book containing a series of simple descriptions of the commoner surgical methods which the plastic surgeon may employ, together with an indication of some of the conditions for which they are likely to be used. The whole is well illustrated by line drawings and photographs, and throughout an indication of the particular activities of the physiotherapist is given. On the other hand, the book will have an equally wide appeal to both nurses and medical students, and it is for this reason that one can almost wish that its title were more indicative of its contents.

R. MOWLEM.

PARASITOLOGY

Introduction to Parasitology. With Special Reference to the Parasites of Man. By Asa C. Chandler, M.S., Ph.D. Ninth edition. (Pp. 799+xiv; illustrated. 68s.) New York: John Wiley and Sons, Inc. London: Chapman and Hall, Ltd. 1955.

Some 35 years ago the first edition of this book was published with the title *Animal Parasites and Human Disease* to present human parasitology in a readable popular form for general readers. It was immediately accepted as an introduction to parasitology for academic purposes; in successive editions, of which this last is the ninth, the scope was widened to include the lower animals as well as man, in order to emphasize the general principles of parasitology that may not be obvious when the parasites of only one host are considered. The approach is primarily biological, stressing the importance of the parasites, their life-cycle, epidemiology, pathogenicity, immunology, and the applied aspects of prevention and control. Taxonomy is considered only so far as is necessary to give an understanding of the general relationships of the parasites. The book is more concerned with fundamental principles than with the details that would interest a specialist.

A comprehensive and integrated account is given of the relevant fields of protozoology, helminthology, and medical entomology, and this approach is most desirable in giving a general introduction to subjects which are so intimately related. The spirochaetes are included with the protozoa, since some are transmitted by arthropod vectors, and arthropod-borne bacteria, rickettsias, and filterable viruses are considered together. Parasitic diseases that are of increasing current interest in the world, such as leptospirosis, amoebiasis, schistosomiasis, onchocerciasis, and leishmaniasis, are dealt with more fully, and the general sections on immunity, insecticides, etc., have been largely rewritten. The guidance given to the literature on parasitology is particularly valuable.

There are occasional statements with which those intimately concerned with particular subjects may not agree, and since this edition was compiled there have been important advances in onchocerciasis and in infections with *Loa loa, Wuchereria malayi,* and *W. pacifica*; but this is inevitable in a subject which is developing so rapidly. This new edition succeeds even better than the last in presenting parasitology to the novice as a fascinating integrated study of living organisms, and is a book which may be read with new pleasure by the parasitologist.

W. E. KERSHAW.

BRITISH MEDICAL JOURNAL
LONDON
SATURDAY FEBRUARY 16 1957

VIRUSES MADE TO ORDER

The relative simplicity with which the plant viruses can be handled has recently led to outstanding advances in the understanding of their structure, and it is no exaggeration to say that it is now possible to take a virus particle to pieces and put it together again without at the same time ending its existence as an infective entity. Tobacco mosaic virus is an exceptionally favourable object for study, since it can be prepared from infected tobacco plants in large quantities and obtained in crystalline form, a circumstance which enables its structure to be investigated by the powerful method of *x*-ray diffraction. G. Schramm, G. Schumacher, and W. Zillig[1] showed that the rod-like particles of virus could be broken down into sub-units by treatment with alkali, and that the sub-units could then be recombined into particles of protein which were of the same size and shape as the original virus, although they were no longer infectious. The sub-units could be further separated from each other by electrophoresis to yield a number of distinct fractions, and when these fractions were examined under the electron microscope an odd assortment of objects came to light. There were virus rods with one or more sections missing out of their middles, and held together by a central filament which evidently ran throughout the whole length of the rod. The missing bits were also in evidence as disks with holes in the middle. From studies of the chemical composition of the virus it seemed probable that the disks were made of protein and were strung upon a central filament of ribonucleic acid. Now protein and nucleic acid are chemical substances of distinctive properties which can be separated and treated by standard chemical procedures, and H. Fraenkel-Conrat and R. C. Williams[2] have been able to prepare each component in a fairly pure state. The protein was precipitated with ammonium sulphate and the ribonucleic acid was obtained by treating the virus with detergent; after purification the protein and the ribonucleic acid were obtained as separate solutions. No trace now remained of the structure of the original virus rods, and the two solutions were not infectious when rubbed on to the leaves of tobacco plants. But when the solutions were mixed an opalescence gradually developed on standing; examination in the electron microscope revealed that rods had reappeared, and that they resembled the rods of tobacco mosaic virus in all respects except that they showed greater variation in length. Moreover, when this material was inoculated on to tobacco plants it was found that 3% of the rods were infectious, and produced the distinctive lesions of tobacco mosaic virus.

In further experiments[3] protein of standard tobacco mosaic virus was combined with ribonucleic acid prepared from certain strains of virus which produced distinctive symptoms, and the reconstituted virus was then inoculated on to tobacco plants. In all cases the lesions which resulted were those characteristic of the strain of virus which supplied the ribonucleic acid. In one instance investigated in further detail the progeny virus which developed in the plant was identical with the parent which donated the ribonucleic acid. It is therefore evident that hybrids can be produced *in vitro*, and the experiments further afford an elegant demonstration that the genetic characters of the virus reside in the ribonucleic acid and not in the protein. The practical aspects of the reconstitution process have been studied by B. Commoner and his colleagues,[4] and by paying careful attention to details it was possible to raise the yield of infectious rods to nearly 10%. They also made an observation of far-reaching implications. Ribonucleic acid extracted from normal tobacco mosaic virus could be separated into three fractions by chromatography, and when these fractions were recombined with virus protein and inoculated on to tobacco plants the three types of reconstituted virus particle produced symptoms which differed widely from those characteristic of the normal virus. The genetic behaviour of the virus had evidently been modified by the procedure, and three new viruses had in effect been created in the test-tube.

The experiments reported so far have all been carried out with ribonucleic acid obtained from tobacco mosaic virus itself, but it now appears that the virus protein will also combine with ribonucleic acid from other sources. The biosynthesis of ribonucleic acid has been studied by S. Ochoa and his colleagues,[5] and they have found that the nitrogen-

[1] Schramm, G., Schumacher, G., and Zillig, W., *Nature* (*Lond.*), 1955, **175**, 549
[2] Fraenkel-Conrat, H., and Williams, R. C., *Proc. nat. Acad. Sci.* (*Wash.*), 1955, **41**, 690.
[3] —— *J. Amer. chem. Soc.*, 1956, **78**, 882.
[4] Commoner, B., Lippincott, J. A., Shearer, G. B., Richman, E. E., and Jia-Hsi, W., *Nature* (*Lond.*), 1956, **178**, 767.
[5] Grunberg-Manago, M., Ortiz, P. J., and Ochoa, S., *Biochim. biophys. Acta*, 1956, **20**, 269.
[6] Hart, R. G., and Smith, J. D., *Nature* (*Lond.*), 1956, **178**, 739.
[7] Schwerdt, C. E., and Schaffter, F. L., *Science*, 1955, **122**, 879.
[8] Mattern, C. F., and Dubuy, H. G., ibid., 1956, **123**, 1037.
[9] Kjellen, L., Lagermalm, G., Svedmyr, A., and Thorsson, K. G., *Nature* (*Lond.*), 1955, **175**, 505.

fixing bacterium *Azotobacter vinelandii* contains an enzyme, named by them polynucleotide phosphorylase, which will cause adenosine diphosphate and other nucleotide diphosphates to polymerize into a long chain of nucleotide units. The product may be regarded as a highly artificial form of ribonucleic acid, artificial in that it does not consist of all four types of nucleotide arranged in a sequence of genetic significance. In spite of this it has now been found[6] that the protein of tobacco mosaic virus will combine with this material as well, forming rods which are indistinguishable from those of the virus itself. These rods were not infectious for tobacco plants, but it can be fairly claimed that they represent a close approach to a synthetic virus, which lacks infectivity only because it has been supplied with the wrong key for unlocking the metabolism of the host cell.

What applications could this work have in the field of human and animal virology ? The material is far less favourable to work with, but it has recently been reported that poliomyelitis[7] and Coxsackie[8] viruses can be crystallized, and crystalline arrays of particles have been seen in sections of cells infected with adenoviruses.[9] It is possible that these viruses have a structure as simple as that of tobacco mosaic virus, and the examination of their crystals by *x*-ray diffraction will soon enable this point to be settled. If they in their turn can be separated into components and recombined, the manufacture of synthetic animal viruses *in vitro* may become possible. Many years may pass before this is achieved in practice, but it offers the possibility of obtaining non-pathogenic variants by a controllable procedure, without having to wait for spontaneous mutants to arise. The use of synthetic viruses prepared in this way might greatly advance the prevention of such a disease as poliomyelitis.

NON-ULCER DYSPEPSIA

Biopsy has often helped clinical progress during the past twenty years. The development of needle biopsy of the liver, originally described by P. Iversen and K. Roholm,[1] has led to notable improvements in the diagnosis of hepatic disease and in the interpretation of the pathological changes occurring. Recently renal biopsy, which was introduced by P. Iversen and C. Brun,[2] has been developed in Chicago by R. M. Kark and R. C. Muehrcke[3] into a reliable technique and has already cast light on the diagnosis, pathogenesis, and treatment of renal disorders. Most biopsy studies of the stomach have sprung from the work of I. J. Wood and his colleagues in Melbourne.[4] In 1949 they described a method of obtaining biopsy specimens of gastric mucosa by passing a flexible tube into the stomach, sucking a knuckle of gastric mucosa into a lateral hole in a small metal cylinder at the distal end of the tube, and then cutting it off by means of a cylindrical knife running within the first cylinder. Their studies confirmed the critical importance of chronic atrophic gastritis and mucosal atrophy in pernicious anaemia and its neurological complication, subacute combined degeneration of the cord. Many centres with much experience of the megaloblastic anaemias now include gastric biopsy as part of their routine investigations.

At page 372 of the *Journal* this week Dr. A. Wynn Williams and his colleagues at the West Middlesex Hospital describe the results they obtained from studying gastric biopsy specimens from a large number of patients suffering from so-called non-ulcer dyspepsia. They use this term to describe dyspeptic symptoms related to the intake of food and occurring in the absence of any focal lesion of the stomach or duodenum (such as ulcer or cancer) and of disease elsewhere, such as in the gall-bladder, appendix, and colon. Such patients represent a formidable challenge to physicians. They are very numerous, as may be seen from the fact that F. Avery Jones and H. Pollak[5] found that two out of every five patients with gastric symptoms do not suffer from peptic ulcer, and in the majority of these patients it is impossible to make any firm diagnosis. More recently, S. Lipetz and colleagues[6] have found that patients with non-ulcerative dyspepsia are even more common in an urban general practice than are patients with peptic ulcer.

Wynn Williams and his colleagues divide the histological changes seen in gastric-biopsy specimens into several categories—normal, chronic superficial gastritis, chronic atrophic gastritis, mucosal atrophy, and minor mucosal changes. In company with other workers they find that atrophic gastritis becomes steadily more common with advancing years. By contrast, the proportion of patients showing superficial gastritis and minor mucosal changes stays relatively constant in all age groups ; this finding agrees with that of Wood's group of workers in their latest report.[7] There are two possible explanations for this difference between relatively mild mucosal changes on the one hand and the severe disturbances of chronic atrophic gastritis and mucosal atrophy on the other.

[1] Iversen, P., and Roholm, K., *Acta med. scand.*, 1939, **102**, 1.
[2] —— and Brun, C., *Amer. J. Med.*, 1951, **11**, 324.
[3] Kark, R. M., and Muehrcke, R. C., *Lancet*, 1954, **1**, 1047.
[4] Wood, I. J., Doig, R. K., Motteram, R., and Hughes, A., ibid., 1949, **1**, 18.
[5] Jones, F. A., and Pollak, H., *British Medical Journal*, 1945, **1**, 797.
[6] Lipetz, S., Sklaroff, S. A., and Stein, L., ibid., 1955, **2**, 172.
[7] Joske, R. A., Finckh, E. S., and Wood, I. J., *Quart. J. Med.*, 1955, **24**, 269.
[8] Shallenberger, P. L., De Wan, C. H., Weed, C. B., and Reganis, J. C., *Gastroenterology*, 1950, **16**, 327.
[9] Palmer, E. D., *Medicine (Baltimore)*, 1954, **33**, 199.
[10] Atkins, L., and Benedict, E. B., *New Engl. J. Med.*, 1956, **254**, 641.

One is that the lesser changes are precursors of the more severe, so that the proportion of patients showing the lesser changes stays constant with advancing years because entrants to the group are roughly balanced by subjects who develop the more severe changes. Alternatively, the lesser mucosal changes can be regarded as not being a significant departure from normality. This does not seem likely in view of the finding that the lesser mucosal abnormalities are associated with an appreciable diminution in the secretory power of the stomach, this group of cases lying intermediate between the "normal" group and those with atrophic gastritis which show severe loss of secretory power.

Wynn Williams and his colleagues have not been able to attach any specific significance to the various mucosal changes met with on gastric biopsy. At the moment, therefore, this procedure does not seem likely to enter into routine medical practice for the investigation of dyspepsia. It has become plain that patients with a normal specimen obtained at gastric biopsy can suffer from non-ulcer dyspepsia, and it is equally plain that a patient's stomach may be the seat of chronic atrophic gastritis without his having a single gastric symptom. Moreover, combined gastroscopic and biopsy studies have shown some startling discrepancies.[8-10] The condition of chronic hypertrophic gastritis, once confidently diagnosed at a glance by gastroscopists, is found to yield a specimen of normal mucosa more often than not. In other words, the type of gastritis most prone to be associated with dyspeptic symptoms turns out not to be "gastritis" after all. The fact that precise clinical significance cannot yet be assigned to the varieties of gastritis in the causation of dyspepsia does not vitiate the value of making careful observations on the state of the gastric mucosa in disease. It is to be hoped that the West Middlesex Hospital group will be able to observe the future progress of many of their patients with superficial gastritis and minor mucosal changes to see if these changes evolve in time into the more profound disturbance of atrophic gastritis and mucosal atrophy, which, if generalized throughout the stomach, is the primary lesion in pernicious anaemia.

BOXING INJURIES

That boxing is by no means the innocuous sport that some suppose it to be is apparent from the attention paid in recent years to the injuries sustained in the ring by both amateurs and professionals. The subject has previously received attention in these columns,[1] and four papers in the present issue refer to various aspects of the problem. Dr. Macdonald Critchley gives a comprehensive review of the subject, illustrating his account with numerous case histories; Drs. J. L. Blonstein and E. Clarke discuss the ill-effects observed in 29 amateur boxers who were severely concussed or knocked out more than once in a seven-month period; Professor M. Paul describes a fatal injury in a young amateur boxer; and Mr. R. Salisbury Woods reports a curious case of dislocation of the shoulder-joint. Boxing enthusiasts have made much of statistics which suggest that the incidence of serious injury is less than in many other sports such as football and skiing; on the other hand, boxing is unique in that the intention of the boxer is to damage his opponent and, if possible, to render him unconscious. Such superficial signs of injury as the flattened nose, the cauliflower ear, and the scarred, hypertrophied eyebrows are insignia of the boxer's trade. Serious damage to muscles and peripheral nerves is a rare occurrence, and so also is fatal cardiac contusion. The local effects of foul blows (the "rabbit" punch to the nape of the neck, testicular bruising from a punch "below the belt," and renal haemorrhage from the kidney punch) have occasionally to be treated, but it is the injuries which may result in damage to intracranial structures and to the eye which constitute a serious threat to life and livelihood.

Fatalities resulting from injuries in the ring, though uncommon, are being reported more frequently, and Critchley remarks that over 200 had been described up to 1950. Most of these have been attributed to intracranial haemorrhage, usually in the subdural space, but it is remarkable how often surgical evacuation of a subdural haematoma has failed to save the patient's life. In Paul's case, such a collection was found and evacuated, but the patient showed severe decerebrate rigidity from the moment of injury and it seemed probable that mid-brain damage had occurred, even though no definite histological changes were found in this region. As E. Jokl[2] has pointed out, the ordinary knock-out may be due to a transient effect of the blow upon the brain stem, and irreversible damage to this area, without striking histological change, is possibly responsible for some fatalities. It should also be remembered that death may result from the secondary head injury which results when the boxer's head strikes the floor.[3]

The punch-drunk state, so graphically described by Critchley in his paper, has long been recognized as a serious cause of progressive physical and mental

[1] *British Medical Journal*, 1954, **2**, 1535.
[2] Jokl, E., *The Medical Aspects of Boxing*, 1941, Pretoria.
[3] Kaplan, H. A., and Browder, J., *J. Amer. med. Ass.*, 1954, **156**, 1138.
[4] Brandenburg, W., and Hallervorden, J., *Arch. path. Anat.*, 1954, **325**, 680.
[5] Doggart, J. H., *Arch. Ophthal. N.Y.*, 1955, **54**, 161.

deterioration in professional boxers whose standard of performance has fallen off, resulting in increasingly frequent knock-outs. This condition of traumatic encephalopathy is often unhappily progressive even after the subject has abandoned the sport; it has previously been attributed to multiple petechial haemorrhages in the brain, but recent histological studies[4] have revealed a diffuse cerebral atrophy with changes like those of presenile dementia. Euphoric dementia with dysarthria and ataxia is seen in the fully developed case of punch-drunkenness, or there may be extrapyramidal manifestations of Parkinsonian type. As Critchley points out, the "groggy state," in which the boxer, "out on his feet" during the contest, is subsequently confused and amnesic, may become increasingly severe after successive bouts and may be a prelude to punch-drunkenness. Blonstein and Clarke assert that in their experience the punch-drunk syndrome has never been observed in amateur boxers, but Critchley reports an amateur champion who developed its characteristic signs. While it is clearly true that the risks of amateur boxing are slight compared with those of the professional sport, the prolonged amnesia which Blonstein and Clark describe in some of their cases clearly differs only in degree from the "groggy state."

The many and varied eye injuries which may occur in the boxer have been comprehensively reviewed by J. H. Doggart.[5] Among the most important are corneal trauma, scleral rupture, tears of the iris, iridocyclitis, cataract, subluxation of the lens, vitreous haemorrhage leading to retinitis proliferans, and retinal detachment which is rarely amenable to surgery. Doggart also describes a patchy choroidal degeneration ("cauliflower choroids"), as well as orbital fractures, glaucoma, and direct damage to the optic nerve. Many of these lesions represent a serious threat to vision. In fact, the list of possible injuries in boxing is a formidable one, but the medical authorities supervising both professional and amateur boxing are advising many sensible precautions in an attempt to limit their occurrence. Serial E.E.G. recordings can be of value in detecting early evidence of cerebral damage, but frequent clinical review by experienced observers, with particular attention to the occurrence and duration of amnesia, may be equally satisfactory. Although much evidence exists to justify the opinion expressed by Blonstein and Clark that amateur boxing in particular is remarkably safe, occasional catastrophes will certainly continue to occur, and it is important that all concerned with the sport, and particularly the boxers themselves, should be aware of the risks involved. Many will feel that the benefits derived from boxing outweigh its dangers, but this opinion will be hard to sustain unless every reasonable step is taken to minimize the risks of permanent disability, particularly that resulting from injury to the brain or eye.

BILIRUBIN

Forty years ago van den Bergh[1] showed that gall-bladder bile and serum from patients with obstructive jaundice reacted directly with freshly prepared, diazotized sulphanilic acid to give an intense purple colour, whereas serum from normal subjects and from patients with haemolytic jaundice reacted only after the addition of alcohol—that is, indirectly. It appeared that the bilirubin formed by the breakdown of haemoglobin was altered in some way by its passage through the liver, but until recently no satisfactory explanation has been forthcoming to explain the van den Bergh reaction. Perhaps the most popular theory was that the indirect-reacting pigment was bound to protein, from which it was released by the addition of alcohol, while the direct-reacting pigment was free bilirubin. Electrophoretic studies, however, showed that both pigments migrate with the serum proteins, and are probably lightly attached to the albumin fraction. A recent detailed analysis[2] has finally disposed of the view that different bilirubin-protein complexes are responsible for the two reactions. An alternative theory, that a chemical change takes place in bilirubin during its passage through the liver, remained unproved, and it was suggested that the liver produces a catalyst which is responsible for the rapidity of the direct reaction.

The pioneer work of P. G. Cole and G. H. Lathe,[3] using the technique of reverse-phase chromatography, has finally settled the question whether there are one or two bilirubins. They found it possible to separate two distinct pigments—a fast moving, water-soluble substance which gives the direct reaction, and a more slowly moving, indirect-reacting pigment, which is identical with crystalline bilirubin. Later work has shown that the direct reacting pigment is made up of two distinct components.[4] Unfortunately these are extremely unstable, a fact which has hindered their identification. Now two papers have appeared almost simultaneously, one from England and the other from the U.S.A., which provide an important clue to their probable nature.[5 6] Chromatographic analysis of the more stable azo-pigments reveals that the direct-reacting bilirubins are glucuronides, which can be converted to bilirubin (indirect-reacting pigment) by treatment with alkalis or with β-glucuronidase. This is a discovery of great general interest. It is well known that many substances are conjugated on their passage through the liver. This has been termed "detoxication" because it applies

[1] van den Bergh, A. A. H., and Müller, P., *Biochem. Z.*, 1916, **77**, 90.
[2] Klatskin, G., and Bungards, L., *J. clin. Invest.*, 1956, **35**, 537.
[3] Cole, P. G., and Lathe, G. H., *J. clin. Path.*, 1953, **6**, 99.
[4] —— —— and Billing, B. H., *Biochem. J.*, 1954, **57**, 514.
[5] Billing, B. H., and Lathe, G. H., ibid., 1956, **63**, 6P.
[6] Schmid, R., *Science*, 1956, **124**, 76.
[7] White, A., Handler, P., Smith, E. L., and Stetten, D., *Principles of Biochemistry*, 1954, p. 64, New York.
[8] Jirsa, M., Veterek, B., and Ledvina, M., *Nature* (*Lond.*), 1956, **177**, 895.
[9] Watson, C. J., *Ann. intern. Med.*, 1956, **45**, 351.

particularly to toxic materials, but there is good evidence that naturally occurring substances—for example, certain steroids—are treated in the same way. Bilirubin appears to be a further example. A. White and colleagues[7] state that "the enhanced solubility in water incident to glucuronide formation . . . may well render them [conjugated substances] more readily dispensable by the body." This would explain the classical clinical observation that bilirubin appears in the urine in obstructive but not in haemolytic jaundice. Glucuronides are not the only compounds available for conjugation, and there may well be other types of bilirubin complex. For example, bile acids are readily demonstrable in the direct-reacting material, and the capacity of bilirubin to form complexes with taurine has recently been reported.[8]

It looks as though a satisfactory explanation of the van den Bergh reaction is available at last. While of great theoretical interest, the clinician may well ask if these findings have any practical value in the elucidation of jaundice. It is probably fair to say that at present the test is rarely helpful, because there are simpler ways of differentiating obstructive from haemolytic jaundice. In parenchymatous jaundice, for which a reliable test is badly needed, the reaction is complex and often equivocal, though if the speed with which the colour develops is taken into account it may be useful in some cases.[9] It seems possible, however, that a study of the bilirubin complexes in disorders in which the liver cell is damaged may yield data which could be applied to the diagnostic problems of the clinician.

DOCTORS' DISEASES

Some data showing that British medical practitioners may be at special risk of dying from acute poliomyelitis are reported by Dr. R. Doll and Professor A. Bradford Hill at page 372 of the *Journal* this week. The authors have derived them from the death certificates of certain doctors who died in the five years 1951–6. Among 34,494 doctors who had provided particulars for these authors' prospective investigations into the relation between smoking and lung cancer were eight men who, in this five-year period, died from poliomyelitis. While the number is small, it seems to be about ten times as large as it would be if medical men experienced the same death rate from this disease as men in the whole population. The difference is striking, and it does suggest that poliomyelitis can with some certainty be added to the list of diseases to which medical practitioners are unusually susceptible. One of these is coronary heart disease. J. N. Morris and his colleagues[1] showed that middle-aged general practitioners are about twice as likely to die from this cause as are men of comparable age in the population as a whole. In the U.S.A. leukaemia has been shown to be more prevalent among radiologists than other physicians,[2] but evidence in Britain is lacking. Deaths registered in England and Wales in 1950 analysed by social class showed a steep decline in mortality from leukaemia from class 1 (in which doctors are grouped) to class 5 for men as well as women.[3] But unfortunately, though we are more than half-way to the next census, no full occupational mortality tables based on the 1951 census have yet appeared. Some recent figures from the U.S.A. show a relatively high death rate for diabetes mellitus among doctors,[4] though their significance to the British scene is debatable.

In seeking an explanation for the unusually high number of deaths from poliomyelitis in their sample of medical practitioners Doll and Bradford Hill mention the belief that people in the higher social classes may be more likely than people in the lower to contract the disease. As they say, no statistical evidence in this country supports it. Nor does living in a healthy environment seem to raise the risk of contracting the disease, as has been suggested, for Bradford Hill and W. J. Martin[5] found no correlation between infant mortality rates and incidence of poliomyelitis in England and Wales. Possibly the clue lies in the inescapable obligations that so often fall to the medical man to fulfil. Doctors may contract the disease no more often than laymen, but, because they keep working too long, pay the penalty of succumbing to a more severe attack.

[1] Morris, J. N., Heady, J. A., and Barley, R. G., *British Medical Journal*, 1952, 1, 503.
[2] See ibid., 1953, 1, 444.
[3] *Registrar-General's Decennial Supplement, England and Wales, 1951, Occupational Mortality*, Part 1, 1954. H.M.S.O.
[4] Dickinson, F. G., and Martin, L. W., *J. Amer. med. Ass.*, 1955, **158**, 1462.
[5] Hill, A. B., and Martin, W. J., *British Medical Journal*, 1949, **2**, 357.

HEALTH SERVICE SUPPLEMENTARIES

There is nothing new in a supplementary estimate for the cost of the Health Service being presented to Parliament; in fact, it has been the exception rather than the rule for Ministers to keep within their original estimates. There are two main reasons for this. One is that in a period of rising prices the spending authorities are faced throughout the year both with a bigger outlay on goods and with claims for higher pay from those providing services. The second reason is that no other department of state, in estimating its expenditure, has to cope with such an unpredictable and uncontrollable demand for its service as has the Ministry of Health. In the early years of the Health Service it was the unexpected demand that made nonsense of the original estimates of the Service's cost. In recent years it has mainly been the need to pay higher wages and salaries that has raised the estimates. Both factors have led to the supplementary estimate for 1956–7, which was presented last week, though increased remuneration is dominant.

The net total of the supplementary estimate (that is, after allowing for an increase in superannuation contributions and savings in certain items such as the poliomyelitis vaccination scheme) is nearly £27m. for Great Britain. All the main branches of the Service share in this increase:

	Extra Amount Needed £		£
Hospitals ..	15,008,000	Pharmaceutical service ..	8,089,000
Local health authorities ..	1,227,000	Dental service	1,635,000
General medical services ..	2,946,000	Supplementary ophthalmic service ..	801,000

The hospitals, as is only to be expected, account for over half the net total of the supplementary, and in fact a supplementary estimate was heralded at the very beginning of the financial year with the announcement last April of new scales of pay for nurses, including mental nurses, and midwives. Since then there have been Whitley awards to ancillary workers and to administrative and clerical staff. For England and Wales these awards, costing an extra £4½m. a year for nurses and about the same amount for the ancillary staffs, account for about £10m. of the extra £13½m. needed to maintain the hospitals in this financial year. The balance is made up by higher prices for goods consumed, such as food and fuel, which account for £2½m., and by an extra £1m. which the hospitals have to pay in rates as a result of the revaluation of their premises.

The supplementary estimate of nearly £3m. in the cost of general medical services mainly represents the extra amount due to general practitioners as a result of the increase in their expenses. The remainder is accounted for by more payments into the central pool for maternity services, the treatment of temporary residents, and so on. Higher remuneration, especially of nurses, is responsible for almost all the increased cost of the local health services. In the remaining branches of the Service it is a bigger demand from the public that is the chief reason for their cost being higher than was originally estimated. This increased demand is reflected most clearly in the extra sums needed for drugs. Under a new agreement chemists are made a slightly bigger allowance for the cost of containers, but the bulk of the additional £8m. needed is to meet an increase in the number and average cost of prescriptions. An even larger supplementary bill for drugs would have been presented had it not been for the change in the prescriptions charge from a shilling a form to a shilling an item, which is estimated to save £1¼m. this year. For some reason the cost of prescribing per head of the population seems to have been more stable in the past year north of the Border than in England and Wales.

COMPOUND ASPIRIN AND STEROID TABLETS

Since the Ministry of Health announced the availability to general practitioners of the newer cortisone analogues prednisone and prednisolone some pharmaceutical manufacturers have been drawing the attention of doctors to the supposed advantages of tablets containing a combination of one or other of these substances with aspirin in a fixed dosage. Advertisements have stated that the combination has a greater anti-inflammatory action than the steroid alone. There is no proof whatsoever of this. In the early days of cortisone investigation claims were put forward that such substances as insulin and *p*-aminobenzoic acid were "cortisone sparers," but these claims were not upheld, and so far no substance has been found to enhance the action of the older or newer steroids.

In one advertising leaflet the indications for the combined tablet are given as rheumatoid arthritis, osteoarthritis, gouty arthritis, bursitis, tenosynovitis, myositis, fibrositis, and neuritis. There is little convincing evidence that oral steroid therapy is of any value in osteoarthritis or in the non-articular varieties of rheumatic disease which involve bursae, tendons, muscles, or nerves. As steroid therapy causes adrenal involution it should be used only where there is a clear indication of its value. A few reports have shown that cortisone or the newer analogues may be helpful in attacks of gout where colchicine is contraindicated, but their main value in the field of rheumatic diseases is in severe active rheumatoid arthritis. Controlled trials by the Joint Committee of the Medical Research Council and Nuffield Foundation[1] and by the Empire Rheumatism Council[2] have shown that in the average case of rheumatoid arthritis steroid therapy is unnecessary, since progress is as good with aspirin. In severe cases, when other methods of treatment have failed to halt the progress of the disease, W. S. C. Copeman and his colleagues[3] have shown the value of long-term treatment with cortisone under careful medical supervision. W. S. C. Copeman and O. Savage[4] have estimated that only 10% to 20% of cases of rheumatoid arthritis should be considered for steroid therapy.

There would seem to be no advantages and some disadvantages, as Dr. F. Dudley Hart has pointed out in a letter at p. 398 in this issue, in combining 0.5 mg. of prednisolone with 5 grains (0.32 g.) of aspirin. For mild cases of rheumatoid arthritis 60 grains (4 g.) of aspirin is an average dose, and this would provide only 6 mg. of steroid—an amount unlikely to have any suppressive effect on the arthritis. In a severe case requiring steroids the average maintenance dose of prednisone or prednisolone is 15 mg. per day, and if that were given by the combined tablet the patient would have to take 30 tablets and 150 grains (10 g.) of aspirin a day. In other words, such tablets, in spite of being the most expensive form of aspirin yet devised, contain steroids in amounts which are quite inadequate for effective treatment.

[1] *British Medical Journal*, 1955, **2**, 695.
[2] *Ann. rheum. Dis.*, 1955, **14**, 353.
[3] Copeman, W. S. C., Savage, O., Dodds, E. C., Glyn, J. H. H., and Fearnley, M. E., *British Medical Journal*, 1954, **1**, 1109.
[4] —— and Savage, O., *Fourth Annual Report on Stress*, 1954, Acta Inc., Montreal, p. 153.

HUNGARIAN MEDICAL REFUGEES

A special fund is being opened to help those medical men and women who have recently had to flee from Hungary. This week in a letter at p. 398 the Chairman of Council of the B.M.A. earnestly solicits the help of all doctors on behalf of these unfortunate colleagues, of whom about sixty have reached Great Britain, while many more are in Austria. He suggests that doctors may if they wish earmark their contribution to be used in Austria; for the medical profession there, through the Austrian Medical Association, has already made great sacrifices to help the refugees. A certain amount of assistance has already been provided from B.M.A. funds,[5] but all that doctors individually can give will be most gratefully received.

[5] *British Medical Journal*, 1957, **1**, 220.

FATAL HOSPITAL FALLS

BY

GAVIN THURSTON, M.R.C.P., D.C.H.

Barrister-at-Law; H.M. Coroner, County of London (Western District)

The incidence of fatal hospital falls is not high, but the matter deserves notice from the standpoint both of prevention and of the risk of litigation. These aspects are, of course, interdependent. The kind of accident caused by falls on polished floors is well recognized and is not dealt with here. This article is concerned with falls, often suicidal, from windows and roofs.

In six months in the West London Coroner's district six such falls have occurred. The area includes 40 hospitals with roughly 13,800 beds. Postulating an 80% bed occupancy and an average length of stay of 20 days per patient, this gives an annual population at risk of about 100,000. The risk of a fatal fall from a window is therefore about 1 in 8,000. Not all the cases are overtly suicidal, but it is of interest to compare this figure with the current suicide rate of 1 in 10,000 in the general population. The six falls occurring during the six months under review seem to represent an unusually high incidence. Against the factor of closer supervision must be set the nature of the hospital population, consisting as it does of sick people who may be temporarily upset by pain, toxaemia, or biochemical disturbances, to say nothing of psychological troubles. During this time no fatal fall occurred among those occupying the 4,000 beds for mental patients.

Case Reports

Case 1.—Two days after an abdomino-perineal resection of the rectum for carcinoma a man aged 66 climbed out of bed at 6.30 a.m., scaled a radiator, and got out of a narrow opening in a window. He fell to the ground and died of multiple injuries. He had been somewhat mentally confused during the preceding night, but not unduly so for an elderly man recovering from a severe operation. The nurses on duty, quite reasonably, did not consider the possibility of such an athletic action, which was carried out under their eyes with such speed that they could not stop him. He was in an open ward. In view of his disordered mental state it was doubtful whether the conscious intention of self-destruction existed, and an open verdict was returned.

Case 2.—A male hospital employee aged 63, while convalescent from cholecystectomy for gall-stones, developed functional retention of urine, for which he had an indwelling catheter. He was distressed by this. At 12.30 a.m. he was missed from his bed and was found to have made his way to a staircase and thrown himself from a window on the floor above the private room which he had occupied. It was clear that he had deliberately killed himself, and the verdict was in accordance with this.

Case 3.—A woman aged 54, who had had bilateral adrenalectomy for carcinomatosis from carcinoma of the breast, was in hospital, in a private room, to have her cortisone dosage balanced. She had had much distressing treatment and knew she had carcinoma. Superficially she did not seem to have been unbalanced mentally. She had had several attacks in which she had become giddy and had staggered while walking. At 3.30 p.m. she fell from a high-silled window just outside her room door. In view of a history of breathlessness and the giddy attacks it was not possible to exclude accident, and an open verdict was returned.

Case 4.—A woman aged 41 was suffering from alcoholism and had had several epileptiform fits, for which she was being investigated in hospital. She was in a general ward and was described as being rather euphoric and facetious and confused at night. Treatment consisted of large doses of vitamin B, paraldehyde, 5 ml. six-hourly, and phenobarbitone, 3 gr. (0.2 g.) intramuscularly if required. During her third night in hospital she seemed more confused than usual and announced her intention of going home. An extra nurse was detailed to look after her. Early in the morning she got out of bed, gathered her belongings, and said she was going to call a taxi. Despite remonstrance she went down the stairs, accompanied by the nurses, who were still remonstrating with her; suddenly she stopped, threatened the nurses with a piece of iron which she had picked up, and slipped through a staircase window on to a flat roof. Still followed by the nurses, she made her way along the roof and climbed a wall at the end. This was thought to be at ground level, but was in fact some distance above a yard. She fell and was killed. The verdict was accidental death.

Case 5.—A man aged 22 with Crohn's disease had been in various hospitals for several months and had had extensive resection of the ileum. During the course of the illness he developed symptoms of anxiety and depression and was admitted to a mental observation hospital. After a time he was thought fit to return to the general ward for further treatment of his somatic disease. He did not then show signs of mental confusion. At 6.30 a.m. he was seen to go to the lavatory, and bid good morning to one of the nurses quite cheerfully. He was soon missed, and was found lying dead on the pavement several stories below the lavatory window. This window was of the casement variety and so situated that accidental falling was impossible. At the inquest the finding was that he had killed himself.

Case 6.—A man aged 68 had had an operation for implantation of radium seeds for inoperable carcinoma of the pancreas. He had had a previous operation and much medical attention. He was aware of the diagnosis. He had been in hospital 12 days, made no complaint, and suffered no pain. At 6 p.m. he went to the lavatory and threw himself from the window. A note written on the edge of a newspaper was found, making clear his intention. The verdict was that he had killed himself.

Discussion

These six cases show the kind of risk involved. It may be recorded here that the three suicides as found at inquest were the only suicides in hospital during the six-months period. Other methods of committing suicide, such as coal-gas poisoning, drugs, and hanging, are clearly less likely in hospital in view of the practical difficulties, so the window-jump must be regarded as the chief danger. Only a small number of cases are recorded, but they are cited to indicate the risk. Any person with hospital experience could add to them, and no particular hospital seems unduly vulnerable. It is easy to be wise after an event, but reasonable precautions should be taken to avoid this kind of death. The problem should be taken into account in planning the design and layout of new hospitals. No claims were made against the hospitals in which these deaths took place, but it is known that liability has been alleged in similar cases. If a claim is made it would be necessary for the hospital authority to show that a reasonable standard of care had been taken to avoid such incidents. What is reasonable would be assessed by referring to current hospital practice; but this is not inflexible, and would cease to be a guide in the presence of some proved danger. Attention to the following points is suggested.

Perhaps the outstanding feature of these cases is that in no instance was the window-fall expected; and the paucity of such events from mental hospitals, where organization is geared to the ever-present possibility of suicide, indicates the importance of the staff being aware of the risk.

It has been noticed that suicide, including cases of self-destruction that lie on its borderland, is often associated with convalescence from major operations, especially where there has been deprivation of an important viscus. The psychopathology of this is outside the scope of this article. It is suggested that patients who show signs of unusual behaviour in these circumstances, even if it is only slight, should be regarded as possible suicidal risks and be placed in a ward on the ground floor. They certainly should not be left in side wards or private rooms without a special nurse. If a psychiatric opinion has been sought and removal to a mental hospital advised, the usual delay associated with administrative obstacles must be eliminated. It has been noted that the nursing staff can more easily supervise a patient in an open ward; but even this, as shown in Case 1, is no guarantee of security, although the risk is clearly diminished. It is not thought that increase of staffing by night or day would make much difference to fatal falls from windows: the patients in these six cases all fell at a time when ward activity was slight.

Precautions of a structural kind seem more practical, though there would be difficulties where buildings are old and financial resources are slender. It should be impossible to open windows more than 9 in. (23 cm.) at the bottom in upstairs wards, staircases, and corridors. Casement windows could likewise be limited. Where this is not feasible, bars should be fitted to afford at least a temporary obstacle to egress from windows. In the case of lavatories, it should not be possible to bolt the door from the inside. Instead, a large indicator showing "engaged" or "vacant" would be suitable.

In the present state of hospital design and routine it does not seem that any further precautions would be expected by a reasonable person.

Summary

The risk of fatal falls from hospital windows is described and illustrated.

Awareness of the risk is important and should be inculcated in nursing staff.

Simple structural precautions are suggested.

MEDICAL ETHICS IN WARTIME

CODE AND EMBLEM ADOPTED BY W.M.A.

The World Medical Association, at its tenth general assembly last autumn, adopted a "Code of Medical Ethics in Wartime" drafted by a joint committee of the International Red Cross, the International Committee on Military Medicine and Pharmacy, and W.M.A. itself. The World Health Organization was represented on the committee as an observer. W.M.A. also adopted an emblem, recommended by the same committee, for civilian doctors (and others) to wear in wartime. National medical associations are now being asked by W.M.A. to approve and support the code and the emblem. The matter is due to be considered shortly by the B.M.A.

Code of Medical Ethics in Wartime

The provisions of the "Code of Medical Ethics in Wartime" are as follows:

1. Medical ethics in time of war is identical with medical ethics in time of peace, as established in the International Code of Medical Ethics of the World Medical Association. The primary obligation of the doctor is his professional duty; in performing his professional duty the doctor's supreme guide is his conscience.

2. The primary task of the medical profession is to preserve health and save life. Hence it is deemed unethical for doctors to: (*a*) give advice or perform prophylactic, diagnostic, or therapeutic procedures that are not justifiable in the patient's interest; (*b*) weaken the physical or mental strength of a human being without therapeutic justification; (*c*) employ scientific knowledge to imperil health or destroy life.

3. Human experimentation in time of war is governed by the same code as in times of peace; it is strictly forbidden on all persons deprived of their liberty, especially civilian and military prisoners and the population of occupied countries.

4. The forbidding of the medical procedures in points 2 and 3 is mandatory under all circumstances regardless of decisions to the contrary issued by either a *de jure* or a *de facto* authority.

5. In emergencies the doctor must always give the required care impartially and without consideration of sex, race, nationality, religion, political affiliation, or any other criterion. Such medical assistance must be continued as long as necessary.

6. Medical secrecy must be preserved by the doctor in the practice of his profession.

7. Privileges and facilities afforded the doctor must never be used for other than professional purposes.

Rules Governing the Care of Sick and Wounded, Particularly in Time of Conflict

1. Under all circumstances every person, military or civilian, must receive promptly the care he needs without consideration of sex, race, nationality, religion, political affiliation, or any other criterion. Any procedure detrimental to the health, physical or mental integrity of a human being is forbidden unless therapeutically justifiable.

2. In emergencies doctors and associated medical personnel are required to render immediate service to the best of their ability. No distinction shall be made between patients except those justified by medical urgency. Services may be refused if other doctors are rendering their services.

3. The members of medical and auxiliary professions must be granted the protection needed to carry out their professional activities freely. The assistance necessary will be given to them in fulfilling their responsibilities. Free passage will be granted whenever their assistance is required. They will be afforded complete professional independence.

4. The fulfilment of professional duties and responsibilities shall in no circumstance be considered an offence.

5. In fulfilling their professional duties the medical and auxiliary professions will be identified by the distinctive emblem of a red serpent and staff on a white field. The use of this emblem is governed by special regulation.

Medical Emblem

It is clear from the fourth Geneva Conventions and Conferences, states W.M.A., that the protection of the Red Cross emblem applies only to doctors, ancillaries, and medical installations in military organizations. Hence, in time of war, the civilian doctor, his assistants, and civil defence units not under military control are without such protection. The new emblem, suggested by the joint committee and approved by W.M.A., is for them to wear. It consists of a red staff and serpent on a white field. The staff is represented by a vertical line, the serpent by a sinuous line, overlying the vertical line, with two undulations on the left side and one on the right (see diagram).

The ultimate success of this emblem and the Code of Ethics as a guarantee of protection for civilian doctors will largely depend, states W.M.A., on the support they receive from national medical associations.

Dr. W. A. HORNE, medical officer of health of Glasgow, has some remarks to make in his Annual Report for the year 1955 about infestation with bed bugs. In that year 15,000 houses in rehousing schemes were inspected and evidence of infestation was found in 66 (0.44%). Twenty years before the percentage of infested houses was 9.2. The use by the disinfestation unit of D.D.T. and "gammexane" is said to give every satisfaction in the eradication of this pest. The total number of "apartments" treated for infestation by bugs in 1955 was nearly 3,000.

Correspondence

Because of heavy pressure on our space, correspondents are asked to keep their letters short.

Hungarian Medical Refugees

SIR,—The many problems presented by the Hungarian refugee doctors have been considered by the Council of the Association, and it is with the full approval of the Council that I now appeal to the profession for subscriptions to a fund to provide assistance for them.

A particularly heavy burden has fallen upon Austria, as will be seen from the following letter, recently received from the Austrian Medical Association.

"Dear colleagues,

"You have certainly heard about the events which happened in Hungary, our neighbour-country, and that the people of Austria helped the refugees with all efforts, achieving great sacrifices. You will realize, however, that so small a country as Austria is not able to help alone for long and therefore asked the freedom-loving world for assistance. Also the doctors of Austria, who certainly are not too well off, helped the Hungarian refugees by giving them free medical treatment and by raising large sums for the general relief funds. However, another problem arose affecting the doctors of Austria, and for the solution of which the co-operation of the medical profession is needed. Among the Hungarian refugees there are many doctors, and the Medical Board of Vienna sponsored a collection to provide a gift of 500 Austrian schillings (20 dollars) for every one of our colleagues. However, the number of the Hungarian doctors increased and is still increasing. Up to date (December 28, 1956) 560 Hungarian doctors called at our office and the Medical Board of Vienna had to raise a loan to continue the financial support given to them. However, we are now at the end of our financial means and therefore ask you to help the Hungarian doctors. We are convinced that, once you have heard about the misery of the Hungarian doctors, the willingness to help your colleagues will be in your country as great as in Austria.

"However, *Bis dat qui cito dat!*

"Will you please transmit financial support for the Hungarian doctors to the Österreichische Ärztekammer, Erste Österreichische Sparcasse, Wien 1, Graben 21, Konto No. 889.685.

"Thank you very much for your co-operation in the name of our Hungarian colleagues."

The Council would particularly like to be able to give prompt assistance to the profession in Austria, which has already shown great generosity to the many refugee colleagues now in that country. It is proposed that a part of the money subscribed should be used to relieve distress among the relatively small number of refugee doctors from Hungary who have come to this country—most of them on their way to other parts of the Commonwealth or to America—but subscribers may earmark their contributions for transmission to Austria if they so wish.

I have no doubt that very many doctors have already subscribed to one or another of the general relief funds, but I hope that some may be able and willing to subscribe again to a special fund for medical refugees, and I appeal especially to any who may not yet have contributed to any fund for refugees from Hungary.

Subscriptions should be sent to me at B.M.A. House, Tavistock Square, London, W.C.1, and cheques should be made payable to the "B.M.A. Hungarian Relief Fund."—I am, etc.,

S. WAND,
Chairman of Council.

London, W.C.1.

Prednisone and Prednisolone

SIR,—In view of the fact that prednisone and prednisolone are now generally released to the profession, your excellent leader (January 26, p. 215) on the subject was most timely and instructive. Judging by the many brochures on the subject issued by various drug houses since then it seems necessary to underline certain of the points made in your leader, for, acting on the different instructions given in the brochures, the practitioner may run into real trouble, particularly as regards gastric intolerance. Dyspepsia is common in steroid therapy, and these new delta compounds are no exception; a patient should always be asked regarding previous dyspepsia and gastric investigations before starting therapy. Most clinics using these substances since they became available to hospitals eighteen months ago have seen haemorrhage or perforation at least once.

The dosage level should be considered one-fifth that of cortisone; the number of tablets required daily remains, therefore, the same if one changes a patient from tablets containing 25 mg. of cortisone acetate to tablets containing 5 mg. of prednisone or prednisolone. Much of the improvement seen in the clinical condition on changing a patient from one drug to the other may lie in actual increase of dosage, some workers assuming the substances are effective in a dose ratio of 1:3 or 1:4. We have learnt with cortisone to restrict the dosage to the lowest effective level, and in treating rheumatoid arthritis, for instance, daily dosage of cortisone nowadays practically never exceeds 100 mg. and is usually 75 mg. or lower. The equivalent dosages of prednisone and prednisolone are 20 and 15 mg., and though this dosage can be, and should be, increased in the short-term treatment of emergencies, such as severe status asthmaticus, the daily dose in continued suppressive therapy should be under 20 mg., except in conditions dangerous to life.

It offends the lovers of champagne that the bubbles in that delightful wine should be removed by special whisks before making champagne cocktails; it offends my common sense that prednisone and prednisolone, whose main virtue lies in causing less sodium retention than cortisone, should be recommended in some brochures for the treatment of adrenal deficiency, but only in conjunction with deoxycortone. As stated in your leader, the delta substances, because of their very slight salt-retaining properties, are not suitable for the treatment of cases of Addison's disease or the adrenalectomized subject; cortisone or hydrocortisone are to be preferred here, with, in salt-losing cases, fluorohydrocortisone. There is no particular virtue in medicine in removing one property in manufacture so that a second drug must be administered in practice.

Finally, a word against compound analgesic cum steroid tablets. If a given dose of prednisone or prednisolone is mixed with an analgesic substance in a compound tablet it may easily lead to over- or under-dosage of one or other substance. Aspirin is so readily available and widely used that it seems unnecessary to invent yet another compound tablet.—I am, etc.,

F. DUDLEY HART.

London, S.W.1.

Prednisone and Nasal Allergic Disease

SIR,—During the last few years I have been lucky to have the opportunity of helping to assess the value of corticosteroids in the treatment of allergic disease of the nasal cavities and para-nasal sinuses. In particular, corticotrophin, cortisone, and hydrocortisone both as solids, suspensions, and in free alcohol solution have been used. They were given either systemically, by local spray, or by local injection under reasonable conditions of controlled trial. No real evidence of lasting or even temporary benefit in a majority of the cases has resulted from this work, so that enthusiasm had flagged somewhat by the time prednisone became available for clinical trial in hospital.

It is therefore most encouraging to find preliminary studies showing that prednisone in small systemic doses can be of real benefit. So far, selection of patients has been strictly limited to the very worst and most intractable cases, usually with polypi and unresponsive to all other forms of treatment. Out of 14 patients treated after removal of gross polypi, 12 have been completely relieved of symptoms, many of them during the first two or three days, and in four cases the sense of smell has returned after many years' absence.

Dosage begins with one 5-mg. tablet three times a day and is proportionately reduced over the next month until a minimal effective dose is found. In several, this appears to be only 5 mg. every other day. Relapses occur when the treatment is discontinued, and so far the longest consecutive period of treatment of any patient is four and a half months.

Such treatment is likely to be purely palliative and has been used in only a small number of the most severe cases. But these early clinical results have been so heartening, and the gratitude of the patients so impressive, that I feel they should be recorded.—I am, etc.,

London, W.C.1. HENRY J. SHAW.

Safety Factor in Car Design

SIR,—As we see the results in hospitals, road traffic accidents as a class cause the most serious injuries, have the highest mortality rate, and are associated with more permanent disabilities than any other type of accident. In 1954 such accidents accounted for 5,010 deaths in Great Britain.

The mortality rate from similar accidents in 1954 in the German Federal Republic, France, and Italy (each with populations comparable but lower than our own) were respectively 11,565, 7,539, and 5,281.[1] Undoubtedly there are many factors contributing to these considerably higher figures than our unenviable record. One may well be the popularity in those countries of small low cars of extremely light construction. Since petrol rationing and the resulting increase of such cars in this country, both of foreign and home make, we have already treated many serious and two fatal injuries in users of such vehicles.

Many years of hospital experience have shown[2] that, from the car-users' (safety) angle, car design and solid construction are important factors. Four good wheels, a few feet of steel between the driver and what he hits in head-on collisions, stoutly constructed body work as a protection against side-swipe accidents and overturning, a safe design of steering-wheel and dashboard, splinter-proof glass, and a car at least high enough to give its driver a good view of the road ahead and its crossroads, are all important factors in safety car design.

From the safety angle, the increasing popularity of small, low, and very lightly constructed cars is to be deplored. A saving in petrol may be bought at too high a price.—I am, etc.,

Birmingham. WILLIAM GISSANE.

REFERENCES

1 G. O. Jeffcoate, International Comparisons of Road Accident Statistics in 1954, Road Research Laboratory (unpublished).
2 Gissane, William, *Practitioner*, 1954, **172**, 620.

Tuberculosis in Glasgow

SIR,—Drs. W. F. Tyrrell and J. Smith have written an interesting and valuable epidemiological study in their paper on pulmonary tuberculosis in north Glasgow (*Journal*, December 22, 1956, p. 1451). It should be of interest to all part-time tuberculosis consultants. Among their most striking results was a definite contact history in 77.6% of the male cases and 75.4% of the female cases, and of a familial nature in the high figure of 88.5% of cases. To obtain such figures the intensity of the contact investigations must have been great but worth while. The following paragraph among their conclusions deserves special mention: "This work has given us some general impressions which we feel may be of value to other workers in the field. For example, we find that visits by the chest physician to the homes of new patients give the health visitor a feeling of greater interest and encouragement, with the added advantage that such visits serve to reinforce, and occasionally redirect, her activities to the best advantage. We also found that home visits made in the evening were most productive in eliciting information and in establishing rapport with the members of the household. Moreover, at such times recalcitrant contacts were found to be in a more amenable state of mind and willing to discuss the reason for their reluctance to be examined. As our figures show, this reluctance could usually be overcome as a result of our methods, which, however, sometimes involved a considerable number of visits."

The authors state, "Overcrowding, poverty, and Irish nationality were found not to be significant factors," and correlate this with Bradbury's[1] well-known, quite different findings in his Tyneside study. Here, surely, like is not being compared with like. In 1932 the country had some three million unemployed, and Jarrow on Tyneside was a real black spot. Visiting that area then was to see real abject poverty, with housing mostly of the "box house" type, with eight rooms and eight families, the staircase turning right and left into complete darkness; the smell of vermin; rat-gnawed waiscoting; male patients in bed spitting on to newspaper; and children swarming about in squalor and dirt. Glasgow in 1956 was not a distressed area, and is not comparable with Jarrow in 1932, either in its economic conditions or the housing.—I am, etc.,

Church Stretton, Salop. G. LISSANT COX.

REFERENCE

1 Bradbury, F. C. S., *Causal Factors in Tuberculosis*, 1933. N.A.P.T.

Incidence of Venereal Disease

SIR,—It is obviously most desirable to know the true incidence of venereal disease in this country, and from this viewpoint the inquiry instituted by the B.M.A. Venereologists Group Committee (*Supplement*, January 12, p. 19) is to be welcomed. However, attention must be called to the statement that there has been a big fall in the number of new cases attending the V.D. clinics. As far as we can ascertain there has been no significant decrease in these numbers, as the average number of new patients attending the clinics in England and Wales in the years 1921–5 was approximately 76,000; in 1934–8, 100,000; and in 1952–5, 97,000. In 1956 the number of cases attending during the first three-quarters of the year totalled 74,000, so that the probable average for the five-year period 1952–6 will be in the region of 98,000.

It is true that early syphilis is rare and late syphilis is also decreasing in most areas, as would be expected. The incidence of gonorrhoea remains disturbingly high, and in some areas, including Birmingham and district, is tending to reach epidemic proportions. This is partly due to immigration, but also, we feel, due to the failure of the medical profession to tackle the problem of gonorrhoea from the aspect of the community as a whole. We feel that many cases of gonorrhoea, and probably a few cases of syphilis, are treated by general practitioners. This they have every right to do, but we wonder if in all cases they follow up the contacts or retrieve defaulters. It is also not generally appreciated that gonorrhoea in the female is frequently symptomless, and that in very many cases there are no significant signs. As a result of this attenuated reaction to infection in females cultural investigation is always necessary in possible contacts.

The above points, we feel, are important factors in the continuing high incidence of gonorrhoea, and practitioners who treat cases of this infection should realize that they have a greater responsibility than that of curing the individual patient.—We are, etc.,

GERALD H. KNIGHT.
W. FOWLER.

Birmingham, 4.

The Hurt Brain

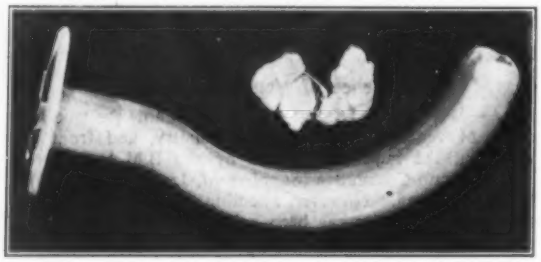
SIR,—In company with millions of viewers, I have watched with interest the series of programmes on the television concerning mental health under the title "The Hurt Mind." I was greatly astonished and considerably disturbed to hear the doctor who presented the series state that syphilis affecting the brain could now be cured with ten injections

of penicillin. Unfortunately, this view seems to be widely accepted in mental hospitals, and yet I feel quite sure that few venereologists would support this statement. It is true that in many patients the symptoms are relieved and greatly improved by such a course of treatment, but that is far from establishing a cure. I fear that these same magic powers are attributed to a short course of penicillin not only by psychiatrists but by many others in the various ancillary branches of medicine, and one cannot too loudly decry this view.—I am, etc.,

Liverpool. E. E. PREBBLE.

Asphyxia due to Faulty Apparatus

SIR,—Having read with interest the letters by Dr. W. N. Rollaston (*Journal*, September 15, 1956, p. 658) and Dr. C. Langton Hewer (*Journal*, September 29, 1956, p. 766) titled "Asphyxia Due to Faulty Apparatus" prompts me to publish the enclosed photograph, which is self-explanatory. Luckily the patient suffered from no ill effects except for some temporary cyanosis.



It looks as if anaesthetists must not only examine the patency of endotracheal tubes and T-pieces but airways as well before they embark on an anaesthetic.

Surely the lesson to be learnt from this photograph is that airways should not be cleaned with pledgets of cotton-wool but by a proper brush on a wire holder.—I am, etc.,

Farnham Royal, Bucks. F. J. RUSSELL STONEHAM.

Treatment of Acute Appendicitis

SIR,—The recent discussion on the conservative treatment of acute appendicitis has occurred at an opportune moment so far as emergency treatment on the high seas is concerned. This week the national dailies have graphically told of the predicament of Captain R. S. Paton, of the *Tahsinia*, who was confronted with a serious case of acute appendicitis in one of his crew. According to the news accounts, he was 700 miles from land and with no hope of skilled help being immediately available. In answer to his messages for advice he was told to operate. Bear in mind this was a ship of 5,680 tons, pitching and rolling in the teeth of an Atlantic gale. There was no anaesthetist and the captain had no medical qualification, but it is alleged he was advised to plunge into the patient's abdomen to look for he knew not what. Wisely he decided to disregard advice, wear ship, and make full speed for land, believing that the circumstances for his first exploratory laparotomy were not propitious.

Following Mr. Eric Coldrey's account (*Journal*, December 22, 1956, p. 1458) of his method of conservative treatment of acute appendicitis, it would seem proper to revise the *Ship Captain's Medical Guide* and to give a detailed method of conservative treatment to be applied in all cases of acute appendicitis when no surgeon is carried aboard. This should enable the patient to be carried in safety to skilled medical help and save the predicament which beset Captain Paton. It is, of course, by no means unknown for captains to embark on surgical treatment of sick or injured members of the crew when no skilled help is at hand. In the days of sail many captains became of necessity physicians, surgeons, and accoucheurs according to the demands of the occasion.

Twenty years ago I sailed in a windjammer whose Finnish mate, Lars Persh, told me of a surgical occasion in his previous ship. He was sailing in the Erikson-owned windjammer, the *Olivebank*, and when about a month from Australia and two months from England a member of the crew had symptoms of acute appendicitis. The captain decided to operate and Persh assisted him. Another member of the crew gave the chloroform. Although he made a good opening in the S.W. corner of the belly (as it is described in the *Ship Captain's Medical Guide*) they failed after prolonged search to find the appendix. The captain sewed everything up in one layer, using fine twine, and the patient recovered sufficiently to walk off the ship some weeks later. My friend, the mate, was well used to doing minor surgery and had plenty of confidence. He would have been quite happy to operate on me had the occasion arisen. On my voyage I had to operate on a septic hand which had gone beyond further conservative treatment, and I sampled some of the minor problems of working in such surroundings. The weather was bad and the ship was in stone ballast, which caused everyone and everything to be thrown from side to side. Nothing would stop still. The patient was held in a bunk while I gave him hexobarbitone and a member of the crew endeavoured to hold me against the bunk for steadiness. Bowls, syringes, etc., rolled in all directions. The lighting was bad. Add to this the need for a laparotomy, a volunteer anaesthetist giving his first chloroform administration, and a lay operator making his first acquaintance with the inside of the peritoneal cavity, and there seems a sound argument for adopting conservative treatment of the acute appendix on the high seas.—I am, etc.,

Sheffield, 8. E. C. ATKINSON.

SIR,—It was with great interest that I read Mr. Eric Coldrey's paper (*Journal*, December 22, 1956, p. 1458) advocating a conservative approach to the treatment of acute appendicitis. I have been engaged in emergency surgery for over twenty years and have performed the operation of appendicectomy, very rarely with drainage, on some thousands of cases at all ages and all stages of the disease. Surely the introduction of antibiotics has not abolished the old surgical dictum, "Where there is pus, let it out." The observation of Professor F. A. R. Stammers (*Journal*, January 26, p. 225) on frozen pelvis as a result of antibiotic therapy with consequent stricture of the rectum should be enough to condemn this method of treatment. I personally consider it dangerous teaching for house-surgeons and registrars to enter the practice of surgery with the idea that in the presence of infection with pus formation antibiotic therapy is the be-all and end-all of treatment.—I am, etc.,

Swansea. J. RUSSELL HUGHES.

SIR,—Your correspondent, Dr. G. L. Alexander, states as follows in his letter (*Journal*, January 5, p. 46): "I seem to remember that during the war a directive was issued by the Admiralty that under no circumstances was appendicectomy to be attempted at sea—the results of keen young chaps operating in destroyers had been so bad." No instruction of this nature was issued by the Medical Director-General and I have no knowledge of any directive of this nature having been issued. I cannot conceive any circumstances in which such an instruction would be sent. As the conditions under which such emergency action would arise cannot be foreseen, a directive on such a matter might have fatal consequences. This is not intended to be an entry into the lists of *pro* and *contra* appendicectomy. Professional freedom as regards treatment in the Royal Naval Medical Service is a much-prized duty and privilege. It would be a great pity if it were to be thought that arbitrary directives are issued governing the conduct of that professional duty.—I am, etc.,

R. C. MAY,
Medical Director-General of the Navy.

London, S.W.1.

Abnormal Haemoglobin

SIR,—I was interested in your review of the abnormal haemoglobin situation (*Journal*, January 5, p. 34). May I be permitted to comment? During a three-month period last year I examined the blood of a number of West Indians and Africans who have recently become resident in S.W. London. The majority were out-patients and not, to my knowledge, first-degree relatives. By electrophoresis and sickling tests the haemoglobin composition of 204 subjects was determined as follows: A, 167 (82%); S, 1 (0.5%); C, 1 (0.5%); A+S, 29 (14%); A+C, 5 (2.5%); S+C, 1 (0.5%); total abnormal haemoglobins, 37 (18%).

These findings confirm that a considerable incidence of abnormal haemoglobins may be found in such a population. It seems important to recognize this fact because the presence of haemoglobin S may be responsible for a wide variety of non-haematological syndromes. For example, in this series (none of whom presented with any haematological symptoms) the patient in whom S only was found by electrophoresis, a 29-year-old housewife and mother of two children, presented with Perthes's disease of the hip. Unfortunately, no family study could be made and we cannot state whether the condition was homozygous or heterozygous, in the latter case coupled with thalassaemia. In sickle-cell thalassaemia the haemoglobin A production of the heterozygote can be suppressed. The combination of sickle-cell trait with thalassaemia is likely in this instance because, in addition to S, there was 36% foetal haemoglobin. Similarly, the combination of S+C haemoglobin was found in a young man who underwent splenectomy for enlarged painful spleen. Both these conditions are presumed to be the result of intravascular sickling, a condition which may give rise to vascular occlusion in almost any organ of the body. Patients may, on account of this, present with such conditions as haematuria, pyrexia, jaundice, abdominal pain, and various neurological lesions. I suggest, therefore, that elucidation of haemoglobin type is required in any anaemia or inexplicable syndrome occurring in patients of West African ancestry.—I am, etc.,

London, S.E.1. J. A. M. AGER.

The Black Sheep

SIR,—Your leading article on Sonne dysentery (*Journal*, January 26, p. 217) draws attention to the likelihood of transmission of infection through the handling by children of infected lavatory seats.

The reputation of the lavatory seat for transmitting disease, like its owner, has had its ups and downs for years, and up to recent times it has been thought that such blame as it has received has resulted largely from its limited scope for hitting back. However, there is now good reason to believe that Sonne dysentery, trichomoniasis, and syphilis through the secondary rash can be transmitted by this means.

The answer to the problem lies in the provision of the squatting type of water closets. These have the following advantages: (1) the feet are the only part of the anatomy to have contact with the lavatory; (2) the lavatory can be cleansed with a water jet from a hose-pipe; (3) absence of upward splash; (4) the squatting position maintains better tone of the abdominal and perineal muscles; (5) more effective evacuation. It may be thought that prejudice against such water closets would make them unacceptable in this country, but nevertheless the two local authorities I serve have provided them in certain of their public conveniences, and a large factory here has adopted this style for its labourers.—I am, etc.,

Dartford, Kent. J. H. HUDSON.

SIR,—May I send you two clues which may help to solve the mystery of the black sheep (*Journal*, January 26, p. 217)? (1) The cheaper lavatory paper now consists largely of what Dr. Samuel Johnson called "interstitial vacuities," through which the faeces can hardly fail to contaminate the right hand. (2) In the days of the high cistern the handle on the dangling chain lay more conveniently accessible to the uncontaminated left hand. The modern cistern, built low and flushed by a button or small lever, is more easily manipulated by the contaminated right hand. Even among the very U, flushing precedes washing.—I am, etc.,

London, W.1. RAYMOND GREENE.

Fatal Reactions to Local Anaesthetics

SIR,—Your annotation on fatal reactions to local anaesthetics (*Journal*, February 2, p. 276) rightly points out the danger of these drugs, especially when applied topically in the dosage normally used. However, as I pointed out in a previous letter (*Journal*, May 28, 1949, p. 957), there appears to be evidence that, in the case of amethocaine as well as cocaine, the real danger is idiosyncrasy rather than overdosage. My experience of toxic reactions has been increased by only one case since the four I mentioned in 1949, but in every one of these convulsions have developed at an early stage in the preparation of the patient for bronchoscopy or laryngoscopy. Case 4, as previously reported, had less than 10 mg. of amethocaine following 1 mg. of cinchocaine, and the highest dosage in the five probably did not exceed 50 mg. when allowance is made for the solution left on swabs or expectorated. No case has occurred late in a difficult bronchoscopy or bronchography in which use of the bronchial spray may almost double the normal dosage of anaesthetics.

Experience here appears to confirm the belief that premedication with a barbiturate reduces the incidence of these reactions. We had nine years' immunity after adopting such a scheme. However, as case 5 proved fatal in spite of thiopentone and artificial respiration with oxygen, we will use lignocaine in future, but will not relax our normal precautions—barbiturate premedication, thiopentone, and a Boyle's machine at hand.—I am, etc.,

Market Drayton, Salop. A. CLARK PENMAN.

Mild Endogenous Depression

SIR,—I was most interested in Dr. C. A. H. Watts's account of the mild endogenous depression (*Journal*, January 5, p. 4). As a retired consultant psychiatrist, I am filled with admiration at Dr. Watts's "clinical insight" into these conditions, and I would like any remarks I may make to be regarded as complementary to his paper rather than critical. I could not agree more with Dr. Watts's contention that these patients must be treated with patience, sympathy, and understanding. But, having said that, instead of "waiting for three months," I would suggest the enormous benefit most of these patients would get from the immediate giving of 4 or 5 E.C.T.s. In the majority of cases there would be no need for the patient to enter a mental hospital. Most mental hospitals now give such treatment freely to out-patients, and in some instances the treatment may be had in the psychiatric out-patient department of a general hospital.

In dealing with any form of depression the adjective mild, in my opinion, is a risky one to use. The most careful examination may miss the depth of the depression, and what may be a mild depression to-day may be a very severe one to-morrow. Therefore, I do not think the practitioner (or psychiatrist) should subject himself to all the worries of the possible outcome of such cases, but should seek the immediate help of E.C.T. (E.C.T. by no means removes the necessity for sympathetic understanding and support—in fact, if given without the latter, it will usually be found to be very unsatisfactory.)

It is difficult to understand what Dr. Watts means by his "anxious patient" who is "rational and he can follow with interest a logical argument" (so, too, can most depressives). If he means uncomplicated anxiety hysteria, then of course we are dealing with an entirely different condition where E.C.T. would be quite valueless, and where, in my opinion, the treatment of choice is suggestion, which, correctly given, should be almost instantaneously effective. But exceeding care is required here in diagnosis, and it should be remembered that the anxiety hysteric, while perhaps full of

fears and anxieties of, as a rule, all sorts of bodily ailments, does not exhibit any of the classical triad (depression, sleeplessness, and tearfulness), either alone or in combination, of depressive states. It cannot, I think, be too strongly borne in mind that the latter symptoms, in any combination, connote a breakdown, and not a "psychological aberration" which will immediately respond to any form of psychotherapy.—I am, etc.,

Northampton. ROBERT THOMPSON.

Neostigmine-resistant Curarization

SIR,—The recent spate of correspondence in your *Journal* on the subject of neostigmine-resistant curarization leaves me bewildered and not a little anxious. As a surgeon, I am wondering whether one can any longer, with a clear conscience, leave the choice of anaesthetic to an anaesthetist. It may be that the time has come to reconsider the advantages and disadvantages of regional analgesia, and to avoid tampering with the patient's vital functions. Each of your correspondents voices a different opinion as to the root cause of his troubles, but the fact remains that several fatalities are admitted, and, unless a solution is forthcoming, many more may be expected. What distresses me most is the lamentable lack of precise description in most of these reports. Your *Journal* reaches every corner of the globe, but of what possible use can such letters be if the cardinal facts are omitted? You, Sir, and your advisers are not blameless in this matter. It is your editorial duty to ensure that the relevant facts are supplied, so that their publication may stimulate healthy and helpful criticism and the high scientific level of your *Journal* may be maintained.

One correspondent reports five cases with extreme vagueness. The notable omissions are: (1) the amount and nature of the relaxant given; (2) the method by which the dose was assessed; (3) whether the drug was given in a single dose or repeated at intervals; (4) whether a CO_2 absorber was used; (5) the proportions of gas and oxygen; (6) blood-pressure readings; and (7) the age, weight, and muscular development of the patient. Given these very pertinent findings, it is at least possible that somewhere in the world a fellow anaesthetist might be able to offer more than a feeble conjecture as to the cause of the condition.

Two other correspondents advance a possible explanation, and submit four case reports, again very incomplete. In the first, the statement is made that the dose of relaxant was not excessive. To my mind, Sir, that bald statement requires supporting evidence. How was the dose assessed? And what was the dose? Further, what proportions of anaesthetic gases were used? Was attention paid to the tidal and minute volumes respired? Was an efficient CO_2-absorber used? What were the findings as to colour, pulse, and blood pressure? Their second case recovered after two hours of artificial respiration, and is summarily dismissed without a single detail, incredible as it may seem, as to the anaesthetic. Such a case might be highly illuminating if fully reported. Their third case report omits to state the proportions of gases used, the amount of relaxant, whether a CO_2-absorber was used, details as to tidal and minute volumes, and the facts as to colour, pulse, and blood pressure. The fourth case report has similar omissions.

It seems to me that such anaesthetists are forgetting their elementary physiology in their search for a scapegoat. I believe that the relaxants are safe if used in the correct dosage; that this dosage should be assessed in the light of many factors, such as age, sex, muscular development, weight, and state of health; that the estimated dose can be easily checked by feeling the resistance offered by the rebreathing bag to manual compression; and, finally, that the reported complications can be avoided by correct dosage, by attention to the fundamental factors of tidal volume, minute volume, and adequate intake and output of oxygen and CO_2 respectively, and not by sanguine reliance on antidotes.—I am, etc.,

Johannesburg, S. Africa. M. ARNOLD.

SIR,—While not wishing to drag out this correspondence, I would like to congratulate your last contributor, Dr. J. A. Scanlon (*Journal*, February 2, p. 283). His point of view savours of common sense and clinical acumen, qualities which are apt to be sadly lacking nowadays. As anaesthetists, we are constantly asked to deal with hopeless cases. The surgical mind appears to be incapable of accepting defeat: modern anaesthesia is apparently innocuous to the patient, and cases are operated on which have already reached a "point of no return." But before that point is reached there is a stage where operation will save life, and it is then that my less spectacular method (*Journal*, December 1, 1956, p. 1303) will prove effective.—I am, etc.,

Glasgow, S.4. ALBERT CHRISTIE.

Translumbar Aortography

SIR,—May I comment on the interesting paper by Mr. H. Gaylis and Dr. J. W. Laws (*Journal*, November 17, 1956, p. 1141) and the subsequent correspondence? As a result of their experimental work Gaylis and Laws advance the theory that aortic dissection from intramural extravasation following translumbar aortography was largely responsible for some of the serious complications previously attributed to toxicity of the various injection media. It is true, as they point out, that there are many instances in which all the contrast medium has been injected into one branch without ill effect and that some of the reported complications have been associated with extravasation. On the other hand, there are just as many instances in which no ill effect has ensued following the extra-aortic or intramural injection of dye, and there are good grounds for accepting the toxic effect of these media on various tissues. Their theory, however, cannot be refuted, and goes far to explain dissection as a factor in some of the complications, but I incline to agree with Dr. David Sutton (*Journal*, January 26, p. 225) that "toxicity of the medium when given in excessive doses is the major factor in such accidents."

At least four factors must be taken into account in the explanation of complications following aortography—namely, technique of puncture, type of medium, quantity of medium, and injection pressure. Dr. Sutton, in his criticism of the technique used by Mr. Gaylis and Dr. Laws, advocates the use of a test dose to ascertain the position of the needle point. While this will reduce the risk of injection into one main branch, I agree with Gaylis and Laws, supported by Lindgren,[1] that it does not completely obviate the possibility of intramural extravasation occurring during the main injection. In an experience of 500 translumbar aortograms performed mainly (95%) for renal diagnosis I have found that when extravasation occurs it is usually as a result of imperfect positioning of the needle before injection. This is largely a matter of technique and should be, to a great extent, avoidable. In practice, the surest method to avoid this mishap is to effect puncture by a needle alone and not by one connected to a closed saline system. In this way the needle may be rotated through a full circle of 360 degrees and adjustment made so that a free pulsatile stream of blood is obtained throughout the whole manœuvre. It is imperative that such a free flow of blood is obtained before injection is made.

As regards the various opaque media, the modern organic iodide compounds, while a great advance on sodium iodide, are not completely free from toxic effect. I insist on a routine intravenous test dose for toxicity and sensitivity, and do not place complete reliance on a previous uneventful excretion urogram in which a different strength if not a different drug may have been used. Sodium acetrizoate 70% ("urokon") has proved, in my hands, the most satisfactory medium so far. I consider large quantities of dye, such as 70–100 ml., to be excessive. The use of more than 20 ml. is no more necessary or desirable in renal angiography than in intravenous urography. In practice I never use more than 18 ml. for renal angiography or aortography, this amount being quite adequate provided reasonably rapid injection is carried out. Further, in the event of extravasa-

tion, it is not unreasonable to assume that the effects will be proportional to the quantity injected.

The most important single factor in the production of good aortograms consists of the rapid introduction of the dye, and there is little doubt that the pictures produced using moderate mechanical pressure are superior to those obtained by hand pressure alone. Dr. Sutton, however, appears to condemn the use of pressure injection and states that such instruments "can be extremely dangerous in other and unskilled hands." This is certainly true, but such a criticism applies to the use of almost every piece of apparatus in the medical armamentarium, and I think the position is fairly summed up in respect of aortography in general by the editorial comment on arteriography[2] that translumbar aortography should be employed "as a diagnostic adjunct by *carefully trained personnel*" (my italics). The use of excessive pressure such as 3,000–4,000 mm. Hg attained by some gas-air machines is to be condemned, but few articles on this subject state the actual pressures used for injection. The lever apparatus and technique which I use have been described elsewhere[3,4] in which 15–18 ml. of dye is injected in one second. This requires a pressure of 800 mm. Hg through a No. 16 S.W.G. needle, which I do not consider excessive compared with a pressure of 500–600 mm. Hg over a period of three seconds required by hand pressure to introduce the same amount. Experiments with this injection pressure showed that the rise in intra-aortic pressure was less than 10 mm. Hg and had no detectable effect on the cardiac cycle. While I may have been fortunate in the absence of any significant complications in this series, I attribute this largely to the small quantity of dye used for injection.—I am, etc.,

Glasgow. W. BARR STIRLING.

REFERENCES

1 Lindgren, E., *Acta Radiol. (Stockh.)*, 1953, **39**, 205.
2 *J. Amer. med. Ass.*, 1952, **148**, 652.
3 Stirling, W. B., *Lancet*, 1955, **2**, 123.
4 —— *Proc. roy. Soc. Med.*, 1955, **48**, 823.

Breasts from Bird-seed

SIR,—The interest which has been shown in the device which I described in my previous letter, "Breasts from Bird-seed" (*Journal*, March 17, 1956, p. 628), indicates the real need which exists for a better form of mammary prosthesis. The purpose of this further communication is to describe an improved pattern, which is now on the market, possessing the mechanical advantages of the bird-seed model but free from its obvious drawbacks.

It may be remembered that the original model consisted of a cloth bag loosely filled with bird-seed, which was inserted into an ordinary brassière. The design of the new one is identical except that "polystyrene" granules are used as a filling instead of bird-seed. The patient who first used the bird-seed model has tried this one out and has found it to be equally satisfactory. It is now going into production, and I understand that a patent has been applied for. It has been given the name of "malpro," and may be obtained, in limited quantities at present, from Eunice A. Morris, 62, Church Street, Blackpool.

The polystyrene granules which are used are similar in size and weight to bird-seed, and so they provide the shifting quality and the exact counterpoise which are needed (see my previous letter). They are non-absorbent and resist boiling, so that washing the prosthesis presents no problem. Although they burn in the open fire, they are not highly inflammable. In conclusion, may I reply to two points which were raised by "Interested Victim" in her letter (*Journal*, April 14, p. 862)? First, although cotton-wool is a satisfactory substitute in ladies of modest proportions, it is quite inadequate in the majority of cases, because it is much too light to retain its place and to counterbalance the other side. Secondly, the discomfort in the immediate post-operative period which she has experienced from the standard models is largely due to their fixed shape and lack of resistance—i.e., a lack of the very shiftiness which has caused such fun.—I am, etc.,

Blackpool. D. K. LENNOX.

Non-united Fracture of Scaphoid

SIR,—In two successive orthopaedic clinics at the R.A.F. Hospital, Nocton, recently, six new cases of established non-union of a fractured carpal scaphoid were referred for diagnosis and treatment. While the number was unusually high it did serve to emphasize the steady stream of such cases encountered. Disability from this lesion is variable, but more often than not manual workers are handicapped by pain, a weakened grip, and restricted mobility, while a skilled craftsman can become seriously disabled. In almost every case the fault lies in premature reassurance of the patient in the surgery or the casualty department as his "sprained wrist" is strapped. This reassurance may have been encouraged by an *x*-ray report of "No bone injury" within a few days of injury. A confirmatory radiograph twelve or fourteen days later (when clinical suspicion should have remained) was not taken.

A non-united fracture of the scaphoid offers considerable difficulties in management. Degenerative osteoarthritic changes are often well established by the time the patient presents for treatment, and there is, unfortunately, no single procedure available that will guarantee union and/or abolish disability in all cases. It is for these reasons, Sir, but above all for the distressingly high incidence of this largely avoidable lesion, that I beg leave to raise this subject in your columns. I realize only too well that this problem has long been widely appreciated. Our experience, however, does seem to underline the value of occasional emphasis on this diagnostic problem.—I am, etc.,

Lincoln. K. R. J. DE BELDER.

Herpes Zoster and Chicken-pox

SIR,—I recently had a case of herpes zoster in one of my patients followed later by chicken-pox in her daughter, aged 21 years. I reviewed the recent comprehensive article on this subject by Dr. R. M. McGregor (*Journal*, January 12, p. 84), and felt that my observations on these cases might strengthen the view that there is some connexion between the two conditions.

The patients in question went away on holiday over Christmas and returned to Liverpool on New Year's Day. Two days later (January 3) the mother developed severe pain in the right hypochondrium radiating through to the back, associated with a feeling of nausea, and this was considered most likely to be an attack of biliary colic. A curious feature is that for three nights the pain recurred at approximately the same time—2 a.m.—although during the day she could bear the pain. I called out a consultant physician on January 4, who agreed that biliary colic was the cause, due to over-indulgence in rich food while on holiday. He suggested I call in a surgeon if there was no improvement in her condition. This I did on January 5, and, although he considered the diagnosis of biliary colic most likely, for the first time it was noticed that she had some vesicles in the region of the umbilicus, in the line of the tenth right intercostal nerve. It was felt advisable to admit her to hospital for Graham's test, which proved negative, and the herpes zoster lesions appeared in typical fashion. After a week she returned home. On January 23 her daughter developed a severe attack of chicken-pox, from which she is convalescing at present.

I feel that in these cases exposure to infection took place during the holiday, and there is presumptive evidence that there is approximately twenty days' difference between the incubation period of chicken-pox and herpes zoster, as both the mother and the daughter must have been exposed to the same source of infection. An interesting feature of the case is that some years ago the mother's father had severe herpes zoster at the same time as his son had chicken-pox. I have seen several cases of herpes zoster in the last few years, but regret that my note-taking has not been as thorough as that of Dr. McGregor, who must be congratulated on his most excellent article. As a point of interest, a recent case of herpes zoster was in a patient suffering from

Hodgkin's disease, and I would welcome any suggestions as to the possible associations between the two conditions.—I am, etc.,

Liverpool, 7. R. H. BRACEY.

SIR,—I was interested in Dr. R. M. McGregor's article on herpes, chicken-pox, and cancer (*Journal*, January 12, p. 84), and since reading it have come across a small outbreak I feel worthy of mention.

The father of a family of grown-up children was diagnosed as having herpes ophthalmicus on January 5. Sixteen days later his married daughter, who lives in the same house, had chicken-pox. Only four days after this her young brother of 17 was similarly diagnosed. A young married woman and frequent visitor of the family, who lived two houses away, contracted chicken-pox on the same day as the daughter. So far as we are aware there have not been any other cases of herpes or chicken-pox in this fairly isolated village for many months. And the patients concerned have had no known contact with these diseases outside their village.—I am, etc.,

Charing, Kent. RAYMOND CASTLE.

Hereditary Factor

SIR,—The report by Dr. Mary E. Carter on two siblings diagnosed as acute lymphatic leukaemia (*Journal*, February 2, p. 265) is particularly interesting to me, because in 1953 I saw a woman of 45 with typical chronic myeloid leukaemia, who had lost identical twins with acute lymphatic leukaemia 20 years earlier. The literature on the possible hereditary factor in leukaemia by Guasch[1] does not contain any similar information, but it may well be that my three cases were coincidental.—I am, etc.,

London, W.1. A. PINEY.

REFERENCE

[1] Guasch, J., *Le Sang*, 1954, **25**, 384.

Pregnancy Sickness

SIR,—I am encouraged to add my mite of personal observation to that of Dr. Doreen S. Ball (*Journal*, December 22, 1956, p. 1487) and Dr. P. G. Cronk and Dr. Muriel Dalby (*Journal*, January 5, p. 49). My medical school is known for its antenatal training, and I anticipated a pregnancy without emesis or toxaemia, with well-being, a "natural" birth, and full breast-feeding, as being the right of a wanted pregnancy and a calm, sensible approach. So eager was I to conceive that I thought myself pregnant before I was, though of course the symptoms did not include sickness. Does pseudocyesis ever include emesis? The first sign of pregnancy was slight giddiness and that a casual throat clearing or cough made me retch at about six weeks. All day nausea and retching attacks developed; flavours went wrong; my mouth seeming to be coated with stale fat, which drove me to eat citrus fruits. Nausea, occasional vomiting, and wrong flavours persisted till delivery. I never felt well and had mild pre-eclamptic toxaemia at 30 weeks.

In labour I had no fear, was completely confident in my doctor and nurses, and had my husband with me. The familiar uterine contractions were painless, but accompanied by a sharp pain in the sacrum, this becoming unbearably severe even with pethidine. The pain had no component of pressure, distension, ache, or drag, and was not like intestinal colic. Relaxation did not ease it. The second stage was comparatively easy. The baby was by my side during the lying-in period, and breast-feeding seemed the easiest and happiest thing, but after two months the supply declined and was not revived by 14 days' religious expressing.

Such experiences open one's mind to try to observe instead of merely applying teaching. Any patient's account published by Dr. Grantly Dick Read and followers describes feeling some very strong sensation during labour, which is not pain. What is it? I am sure that only a few "trained" mothers achieve painless labour (I have not personally known of one), while a few "untrained" mothers have painless labours anyway. Are there any comparative figures? I do not decry antenatal training in the least, but so much depends on the personalities of those who attend women in labour. I have heard a nurse answer an appeal from a distressed woman with, "You've been taught to relax—get on with it," then leaving her alone. It is the absence of fear that is all-important.—I am, etc.,

Emsworth, Hants. ANN PARKER.

Methyltestosterone Therapy

SIR,—In response to the request of Drs. G. L. Foss and S. L. Simpson (*Journal*, December 15, 1956, p. 1426) we wish to report an additional case of jaundice due to methyltestosterone. A female patient, aged 50, diagnosed as a case of hypopituitarism, was admitted to Sheffield Royal Infirmary and treated with methyltestosterone linguets (20 mg. daily) from March, 1953. In July, 1953, she became deeply jaundiced, and three months elapsed before the patient returned to her normal colour. The results of liver-function tests between the onset of jaundice and December, 1953, are tabulated below.

The main abnormalities are a raised serum bilirubin and alkaline phosphatase with a normal thymol turbidity. Two weeks after the jaundice became evident the patient's haemoglobin fell to 7.4 g./100 ml., and, though no frank melaena was observed, the stools contained occult blood at this time.

Date	Thymol Turbidity	Serum Bilirubin (mg./100 ml.)	Serum Alkaline Phosphatase King–Armstrong Units	Serum Albumin (g./100 ml.)	Serum Globulin (g./100 ml.)
1953					
July 16	1		42	4·1	2·2
,, 30	<1	37	37		
Aug. 10	<1	22	23	4·3	2·3
,, 20	<1	13·8	20		
,, 28	<1	10·4	18		
Sept. 2	<1	6·9	17	4·9	3·0
,, 9	<1	5·0	16	4·7	2·3
,, 15	1	3·6	15		
,, 24	1	1·8	11		
Oct. 14	2	2·5	7		
,, 21	2	1·6	12		
Dec. 2	1	0·8	8	5·0	3·1

After transfusion and oral iron the anaemia was corrected, and repeated examination of the stools showed that they contained no occult blood. The interval between starting the drug and the onset of jaundice is in keeping with most of the cases reported by Werner and others,[1] who also found the serum alkaline phosphatase to be raised. The case reported by Wood[2] had a normal serum alkaline phosphatase.

Since recovering from jaundice the patient has been well and treatment with methyltestosterone has not been resumed. The number of reported cases is small, and the complication is so rare that it should not be regarded as a contraindication to using this drug.—We are, etc.,

Manchester. S. OLEESKY.
Sheffield. D. S. MUNRO.

REFERENCES

[1] Werner, S. C., Hanger, F. M., and Kritzler, R. A., *Amer. J. Med.*, 1950, **8**, 325.
[2] Wood, J. C., *J. Amer. med. Ass.*, 1952, **150**, 1484.

Tobacco-cancer Problem

SIR,—If Dr. Lennox Johnston (*Journal*, January 26, p. 228) is correct, as I believe he is, in asserting that tobacco smoking is essentially a manifestation of nicotine addiction, there seems to be a simple solution to the cancer-smoking problem: nicotine could be presented in some bland vehicle providing characteristic and pleasant sensations of taste, flavour, touch, etc. For the edentulous, a flavoured sucrose pastille might be suitable, but for others some non-fermentable (non-cariogenic) substance would be preferable—e.g., sorbitol, as in diabetic sweets, or chicle gum sweetened if necessary

with saccharin. I suggest a sweet or sweetened solid vehicle because those who are giving up smoking often find the use of ordinary or diabetic sweets helpful, but I imagine that some other consistency and taste, or combination of tastes, would be found equally pleasant if it were especially associated with the pleasure derived from nicotine, in the same way that a degree of bitterness that would otherwise be objectionable is pleasant in some drinks containing caffeine or alcohol.

Such a solution to the cancer-smoking problem might possibly lead to an increase in nicotine poisoning, but it surely deserves consideration. The ideal solution, of course, would be the discovery of some perfectly harmless but identically satisfactory substitute for nicotine itself. I doubt if education, propaganda, and legislation will themselves do much to prevent tobacco-smoking. Even middle-aged doctors obviously prefer to run the risk of cancer rather than forgo the pleasure of absorbing nicotine from tobacco smoke. Prohibition could only be accepted, if at all, when every possible effort to find a constructive solution had failed, and this is very far from the case at present.—I am, etc.,

London, S.W.10. R. B. D. STOCKER.

Check the Dose

SIR,—The anticoagulant drug phenindione is familiar under the trade name "dindevan" as scored tablets in one strength only—50 mg.—from which dosage schedules in multiples of 25 mg. may be arranged. There is now a need for a weaker strength if advantage is to be taken of the more accurate stabilization of patients by means of Owren's[1] modified method of estimating the "one-stage prothrombin time." The advantages claimed for this method of controlling anticoagulant therapy are that it is sensitive to dosage increments of 10 mg. and thus it is possible to stabilize patients whose dosage requirements do not coincide with multiples of 25 mg.; trends towards overdosage are obvious and the correct dose can often be decided two or three days earlier than with the older method.

The makers are ready to supply a 10-mg. tablet of dindevan to meet this need. With this new strength, however, difficulties for the prescriber and pharmacist may arise. The *British Pharmacopoeia, 1953—Addendum, 1955*, directs that, "if the quantity [of phenindione] contained in a tablet is not stated, tablets containing, in each, 50 mg. shall be supplied or dispensed." If this directive were followed, the pharmacist would supply tablets which might be five times the strength of those intended.

So that there may be no doubt as to what should be supplied, prescriptions or orders for this drug should state the precise dose in milligrams or the strength of the tablet required.—We are, etc.,

ROSEMARY P. BIGGS.
W. TRILLWOOD.

Oxford.

REFERENCE

[1] Owren, P. A., *Schweiz. med. Wschr.*, 1954, **84**, 822.

Wellcome Floating Laboratory

SIR,—Under the heading "Benefactions by the Wellcome Trust" (*Journal*, February 2, p. 295) there occurs the following statement: " . . . probably the first floating laboratory to be made was the one built in 1907 to the order of the late Sir Henry Wellcome. It was used on the Nile by members of the staff of the Gordon Memorial Hospital, Khartum."

There has, in fact, never been a hospital in Khartum called the Gordon Memorial Hospital. The floating laboratory was given by Sir Henry Wellcome to the Wellcome Tropical Research Laboratories, which he had himself founded in 1902–3. It was first used by the director, the late Sir Andrew Balfour. The floating laboratory was loaned to me for several months in 1915 when I was dealing with a devastating epidemic of cerebrospinal meningitis in the then White Nile Province of the Sudan.—I am, etc.,

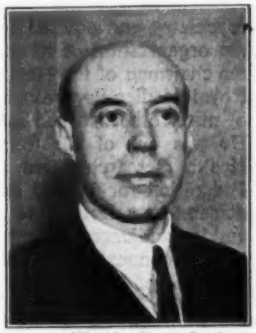
London, W.1. H. C. SQUIRES.

Obituary

D. R. MacCALMAN, M.D., F.R.C.P.Ed.

Professor Douglas MacCalman, formerly Nuffield professor of psychiatry at the University of Leeds, died after a prolonged illness on January 31 at the age of 53.

Douglas Robert MacCalman was born on December 4, 1903, in Strachur, Argyllshire, the son of the Rev. D. M. MacCalman. Brought up in the north-west Highlands, he never lost his deep affection for that remote and lovely part of the country, and the influence of those years was always apparent in his own quiet and courteous manner. He was educated at Glasgow Academy and then at the University of Glasgow, where he graduated M.B., Ch.B. in 1929, winning the Hunter medal in pathology. As with many other sons of the manse who have studied medicine, he was at an early stage in his career attracted to psychological medicine. Soon after qualification he took up appointments in psychiatry in the United States, first at Phipps Clinic, Johns Hopkins Medical School, Baltimore, under the late Professor Adolf Meyer, and then at the Boston Psychopathic Hospital, under the direction of another illustrious Scot, Dr. McFee Campbell, professor of psychiatry at Harvard. Having come under the tutelage of two of the most eminent psychiatric teachers of our time, MacCalman was well fitted to return to this country and assume a position of leadership at an early age. In 1932 he was appointed medical director of the Notre Dame Child Guidance Clinic in Glasgow and assistant physician at the Victoria Infirmary, having in the previous year proceeded to the M.D., with high commendation in psychiatry. In 1935 he was invited to become general secretary of the Child Guidance Council in London, where he enhanced his reputation not only as an able clinician but as a man of outstanding academic and administrative ability.



[*Kemsley Picture Service*

The institution, in 1938, of the Crombie-Ross lectureship in psychopathology at Aberdeen University was an important new development, for it was the intention of the founder and of the university to extend teaching and research in psychological medicine into the general teaching hospital and to enlarge the mental health service by setting up out-patient clinics for adults and children. Dr. MacCalman was the first and only holder of this appointment, which carried with it the honorary status of assistant physician at the Aberdeen Royal Infirmary and the Aberdeen Hospital for Sick Children. Throughout the war years the new department was gradually built up in face of wartime restrictions, shortage of staff, and the like. Additional duties in connexion with the emergency medical services were placed on his shoulders. Despite these great handicaps and the mounting burden of private practice, he succeeded in establishing himself and his subject in the north-east

region with such success that the fruits of his work are still being gathered to-day. It was singularly appropriate that when it was decided to create a professorial department in Aberdeen Dr. MacCalman should become the first incumbent of the chair. In the next three years the unit was extended and the teaching programme completely reorganized, so that now a comprehensive course of over 100 hours is given to the undergraduates during their third to fifth years of study. His opinions were much sought after in the early days of the National Health Service, and his vision contributed greatly to the close integration of the mental health service in the north-east region.

In 1948 he decided to apply his great gifts to a new and larger sphere when appointed the Nuffield professor of psychiatry at the University of Leeds. Here again he built up a university department, both in its teaching and in its research functions. He organized the training scheme for the University of Leeds diploma in psychiatry and advised the regional hospital board on the organization of its mental health service. He was the chairman of the North and Midland Division of the Royal Medico-Psychological Association and a member of many other learned societies and official committees. He was one of the founder members of the Mental Health Research Fund and a Trustee of the World Federation for Mental Health, and he was in constant demand as a lecturer to medical societies, conferences, and lay audiences. One of his outstanding achievements in this connexion was his address to the first World Congress of Mental Health in London in 1948. He was invited to lecture in Finland, the United States, and more recently in Australia, and wherever he went his sterling gifts were apparent. In his early years as a psychiatrist he took part in a large-scale scheme of research into the causation of schizophrenia, and he also made a study of the Rorschach test. He contributed chapters to a number of books and published articles on child psychiatry, education, and neuroses. At the Annual Meeting of the B.M.A. in 1953 he served as vice-president of the Section of Psychiatry. He was elected to the Membership of the Royal College of Physicians of Edinburgh in 1948 and to the Fellowship in 1952.

In recent years ill-health began to impair his prodigious energy, but characteristically he carried on doggedly and without complaint. Though often in considerable pain, he never made reference to it. After a mild thrombotic episode, he eventually decided that he must retire from his chair, but continued in active work, both clinical and academic, almost to the end.

In his younger days MacCalman was an all-round sportsman. He was a fast and elusive wing three-quarter for Glasgow Academicals, and played low-handicap golf until arthritis made this impossible. He had a lifelong interest in gardening, ornithology, and painting, liking nothing better than a painting holiday in Ullapool. An ardent exhibitor of fruit and vegetables at local flower shows, he was always greatly delighted when he won a prize for his products. He was extremely well read, not only in his own subject but in English literature and history.

Professor MacCalman married Helen, only daughter of Dr. J. M. Dawson, in 1935, and she survives him, with one daughter.—W. M. M.

G. R. H. writes: As the first holder of the Crombie-Ross chair of mental health at Aberdeen Douglas MacCalman combined his earlier experience in the mental hygiene of childhood with his developing interest in the teaching of medical students. This combination of interests he continued to pursue when at Leeds. The attention which the Leeds medical school pays to psychiatry in both its undergraduate curriculum and its final examination bears witness to MacCalman's personal conviction of the importance of this subject in the family doctor's daily work. The existing postgraduate training of psychiatrists at Leeds was continued and developed by MacCalman, and the introduction into the curriculum of the course for the Diploma in Psychological Medicine (Leeds) of teaching on certain aspects of the social sciences and on psychotherapy is evidence of his own broad orientation in psychiatry. Since he had taken up his chair at Leeds in the early days of the National Health Service it was always one of his main aims to strengthen the links between his department in the university medical school, the clinical work of the teaching hospitals, and the psychiatric services of the regional hospital board. Both as a consultant to St. James's Hospital, Leeds, and to the City Council of Leeds, and as chairman of the Mental Health Technical Advisory Committee of the Leeds regional hospital board, he did much to build a closer relationship between the various aspects of the cure and prevention of mental illness in the region.

To those who did not know him well Douglas MacCalman must sometimes have seemed a shy man until his interest in a topic broke through his natural reserve, as it would in discussion, teaching, or public speaking, for which he had a great natural gift. Those who worked with him developed strong bonds of affection with him, and all who saw the way he faced his long and crippling illness admired his great courage. His colleagues and his friends in many countries will feel deep sympathy for his widow and his daughter.

M. B. H. W. writes: I should like to try to express something of my personal concept of and indebtedness to the late Professor Douglas R. MacCalman. In so doing, I am doubtless giving utterance to the thoughts and feelings of the many other psychiatric social workers who have known his encouraging and enabling way with us. Under his aegis as secretary of the Child Guidance Council, a few of us in the 1930's were lent to authorities who wished to start child guidance clinics. We would wish to thank him for his quietly effective support in these ventures and for his sure and careful guidance of our committees. Thanks to his unswerving convictions, out of these small beginnings the present child guidance services have been enabled to grow. In the 1940's, a period of challenge—from without from the rapidly developing mental health services and from within by reason of our growing consciousness of our roles in this field—Professor MacCalman helped many of us individually with our conflicts and problems, never withholding himself when his help was sought. A wider circle will recall his several addresses to the Association of Psychiatric Social Workers: in these he gave full measure of his rich, literary, artistic, humanist self—brilliant and bright—renewing us. It is this quality of renewing, this therapeutic personality, which, as his psychiatric social worker over a period of sixteen years, I feel was his peculiar gift as a psychiatrist; and as I write I recall the many patients, simple and sophisticated alike, who have spoken of this. As a professor, he has been cut off before he could reach his full stature, but for the advancement of psychiatry he imaged the whole; now others must execute the parts. As a colleague he was benign and as a friend he was lovable.

W. H. WILSON, D.M.

Professor W. H. Wilson, emeritus professor of physiology in the University of Cairo, died at the Victoria Hospital, Cairo, on November 14, 1956. He was in his 91st year.

William Hawkins Wilson was born on February 16, 1866, the second son of the late J. C. Wilson, D.C.L., a law tutor at Oxford, and was educated at Radley College

and at Keble College, Oxford, where he was a scholar. In 1888 he obtained a first-class in the Honours School of Physiology and his B.A. degree. From 1888 to 1890 he was science tutor at Keble College and university demonstrator in histology. Deciding to obtain a medical degree, he went on to St. George's Hospital with a scholarship and graduated B.M., B.Ch. in 1893. Before this he had been awarded a Radcliffe travelling fellowship and spent two years studying in Paris, Vienna, Dresden, Munich, and Freiburg in Baden. After qualification he worked as a house-surgeon and house-physician at St. George's. He proceeded to the D.M. in 1903.

In 1895 Wilson was appointed by the Egyptian Government to the post of assistant professor of physiology at the School of Medicine at Kasr-el-Aini, Cairo (afterwards known as the Faculty of Medicine of Cairo University), and he became professor a year or two later, occupying the chair until 1928, when he retired. Later, he was elected emeritus professor of physiology and carried on research work in the physiology department of the faculty and acted as examiner in his subject. He contributed papers on a variety of subjects to the *Journal of Physiology* and other journals, and had made a special study of scorpion venom. During the first world war he remained in Egypt and continued his official duties, also acting as adviser in physiology to the British Army and Air Force. He founded a charitable dispensary at Mataria, Cairo, in 1921, which served a large number of villagers in that area, having a yearly visiting attendance of many thousands. He was a member of the General Board of Health, on which he represented British interests. A member of the British Medical Association for over 50 years, he was president of the Egyptian Branch in 1929–30. He married Kate Hamilton Naylor in 1897 ; she died in 1935.

C. B. S. E. writes: The late Professor W. H. Wilson, who was appointed to the inspectorate of the Department of Public Health in Egypt in 1895, became much interested in the country, more especially as the increasing national budget permitted of extensive developments in the health service, including the foundation of hospitals and dispensaries in every province, and of the enlargement of the teaching hospital of Kasr-el-Aini, in Cairo. He had a natural sympathy and understanding of the most varied classes of the inhabitants, from his nearest neighbour, Prince Youssef Kamel, to the fellaheen workers and small proprietors by whom he was surrounded in Mataria, an outlying suburb of Cairo. There, shortly before the outbreak of the first world war, Professor Wilson and his wife had settled, building a house in a country property ; in this he resided until his final illness. His life there was marred only by a disastrous fall during the building of his home, a fall which resulted in serious spinal and internal injuries, crippling him for life so far as locomotion was concerned, but in no way affecting his mental energy or his determination to improve the life of all around him.

At this period he was appointed professor of physiology at the Kasr-el-Aini Medical School, a post in which he remained until superannuated. He continued, however, to take part in the annual examinations until quite recently, when well over his 80th year. The ailments of advanced age further limited his physical capacities, but did not prevent him from flying to England and back in 1955. During his professorship his personal interest in the students provided him with devoted friends among the Egyptian medical leaders in his later years. He conducted much physiological research at the school laboratories. Until three years ago he was also a member of a dietetic research committee of the Public Health Ministry (formerly Department), which laid down rules for the diet in schools, prisons, hospitals, and for manual workers based on the habits and possible food resources of the country. His close personal touch with working classes living around him, and their womenkind, gave him unusual opportunity to study these matters. In the course of the second world war, when research laboratories were no longer open to him, he invented an instrument to facilitate the sight in night flying, an instrument which was put to use by the R.A.F. He completed the whole work himself in a small wooden hut in his grove of apricot, orange, and almond trees.

His kindness of heart was shown by the fact that on Fridays, his only day free from official work, he opened his doors to the poor and needy in the villages near him, giving them advice, drugs, and money for years. About 36 years ago, with the financial backing of his friend and neighbour, Prince Youssef Kamel, he founded a clinic in Mataria village near his home. In doing this he was supported by some of the best-known medical men in Cairo. He supervised the clinic personally for many years. Fairly recently some official support for it was arranged. Advancing years seriously affected his sight, yet he patiently kept abreast of physiological progress and was ever ready to talk over some new development. During his last days in the Victoria Hospital he had the consolation of visits from several leaders of the medical profession in Cairo, old pupils of his. In spite of the expulsion orders served on the leading members of the English world in Cairo a few days before his death, an English lady was enabled to attend his funeral in company with one or two friends of other communities, including Danish and Egyptian.

H. M. SHELLEY, F.R.F.P.S., M.R.C.P. D.T.M.&H.

Dr. H. M. Shelley, formerly director of medical and health services in Cyprus, died suddenly at sea on January 26 while travelling from Cyprus to Greece. He was 56 years of age and had been ill for some time. He was buried in Athens.

Horace Minton Shelley was born at Bradford on January 11, 1901. From the Intermediate School, Nottingham, he went on to study medicine at Sheffield University. Qualifying in 1923, he joined the Colonial Medical Service in the following year and his first post was in Nyasaland, where he remained until 1939. He took the D.T.M.&H. in 1927 and the M.R.C.P. in 1938, and in 1934 he was elected F.R.F.P.S. of Glasgow. After working for a few years as a pathologist in Nyasaland he transferred to Tanganyika as a senior medical officer in 1939. In 1947 he was appointed director of medical services in Cyprus, where he successfully carried out many reforms and was in charge of the campaign which freed the island from malaria. One of the reasons for the success of this campaign was the friendly relationship which Dr. Shelley established with all his colleagues, medical and administrative. He retired from the Colonial Medical Service in 1952, but continued to live in Cyprus.

An active member of the British Medical Association, Dr. Shelley was honorary secretary of the Nyasaland Branch for six years and president of the Branch from 1934 to 1935. He wrote a small textbook entitled *Epitome of Laboratory Diagnosis and Treatment of Tropical Diseases*, and, having founded the *Cyprus Medical Journal*, he continued to edit it until the time of his death. He married Claire, daughter of Madame Boulenger. She survives him with three children of the marriage.

J. E. G. writes: Dr. Shelley had been ill for several years, and was on his way to seek quieter retirement in South Africa. He had an interesting career, for he was a personality and took a deep interest in many things beside his

work. He boxed as a flyweight for the British against the American universities, and his life was indeed a continual and often successful fight against great odds. He never accepted defeat, and even in a long struggle against a failing heart he seemed to be the attacker. He had more bravery than most men, and rose above his physical incapacity and pain until it mercifully overwhelmed him completely and suddenly. He had dramatic talent and acted on the stage for a time, but mostly this ability was well used in informal medical teaching. Even without his extensive knowledge he would have been impressive. In territories he served in he worked hard and devotedly with the help, and sometimes the handicap, of an imagination not often found in administrators. During his tenure in Cyprus the eradication of the malaria-carrying mosquitoes was completed, and most of this was due to his efforts. He was fond of writing and lecturing, and in addition to his medical writing he wrote fiction and poetry. There are many who will mourn his passing and many whose lives will miss the stimulation of his ideas.

R. W. D. LESLIE, C.B., C.B.E., L.R.C.P.I.

Major-General R. W. D. Leslie, who after a distinguished career in the Army Medical Services, gave valuable help with the organization of the Emergency Medical Service, died at his home at East Leake, near Loughborough, Leicestershire, on January 31, his 74th birthday.

Robert Walter Dickson Leslie was born on January 31, 1883, and was educated at Michaelhouse, Natal, South Africa, receiving his medical training in Dublin. He qualified by taking the Irish Conjoint diploma in 1904. Two years later he entered the R.A.M.C., and when the first world war began in 1914 held the rank of captain. In the twenty years from 1917 to 1937 he rose from the rank of brevet major to that of major-general, holding many important posts in that period. For his services in the first world war, towards the end of which he was an A.D.M.S. in France, he was mentioned in dispatches and appointed O.B.E. in the 1919 Birthday Honours.

Promoted to lieutenant-colonel in 1930, Leslie was deputy assistant director of medical services in India from that year until 1932, and in 1936—having been promoted colonel two years before—he was deputy director of medical services in Palestine and Transjordania. Returning to the United Kingdom, he was in command of the Queen Alexandra Military Hospital, Millbank, until 1937. In that year he became major-general and was appointed deputy director of medical services in Northern Command. He occupied that post until 1941, when he was appointed C.B. and retired from the Army, and from 1939 to 1941 he was an Honorary Physician to the King. During the course of his career in the A.M.S. Leslie contributed a number of articles to the *Journal of the Royal Army Medical Corps*. These included papers on improvements in motor ambulance construction (with Captain G. A. Child); improvised ambulance trains; the medical services in Palestine; and the casualty card scheme in R.A.M.C. field training.

The end of Leslie's military career was by no means the end of his working life. In 1941 he obtained an appointment as a hospital officer in the Emergency Medical Service, and from 1948 to 1954 he was employed as a regional medical officer (No. 3 Region) under the Ministry of Health. For his services in the E.M.S. he was appointed C.B.E. (Civil Division) in the 1946 New Year Honours. Leslie was a member of the Council of the British Medical Association from 1942 to 1948. He also served on the old Naval and Military Committee of the Association from 1944 to 1948 and on the General Medical Council Committee (1946–8) and the Central Medical War Committee (1947–8). He married Miss Eleanor V. Jackson in 1911, and she survives him.

Dr. RUFUS C. THOMAS, consulting obstetrician and gynaecologist to the Kent County Council, died at his home at Croydon on January 11 at the age of 67. Rufus Clifford Thomas was born at Llantwit Vardre, near Pontypridd, in June, 1889, and was educated at Christ College, Brecon, University College, Cardiff, and at the Westminster Hospital. After qualifying M.R.C.S., L.R.C.P. in 1915 he held the appointments of resident obstetric assistant and senior house-surgeon at Westminster Hospital, and then served for a time in the R.A.M.C. From 1919 to 1927 he was in practice at Newport, Monmouthshire, and while there was elected F.R.C.S.Ed. in 1924. At Newport he was also honorary anaesthetist to the Royal Gwent Hospital, consulting surgeon to the maternity and child welfare clinic, and consulting gynaecologist to the borough council. In 1927 he went to India as chief medical officer to the Maharajah Jamsaheb of Nawangar—"Ranji," the famous cricketer. In India his work was partly administrative, but largely surgical. He left India in 1933 because of illness, and joined the staff of the West Middlesex County Hospital as resident surgeon. Two years later he became obstetrician and gynaecologist at the Mayday Hospital, Croydon, and consultant to the county borough council, and in this post his clinical and administrative skill was used to the full. Some two years after the end of the second world war he accepted a similar appointment under the Kent County Council, with headquarters at Farnborough County Hospital. He was a lecturer and examiner for the Central Midwives Board. Elected a Member of Royal College of Obstetricians and Gynaecologists in 1934, he became a Fellow in 1947, and from that year until 1952 served on the council of the College. He was also a member for some years of its Hospital Recognition Committee. He was president of the Association of Whole-time Salaried Specialists. He leaves a widow and a daughter.

K. G. S. writes: Having worked with Rufus Thomas for nearly twenty years, I feel his loss very keenly. In the early days of our association his acumen in diagnosis and strict attention to detail were at once an example and an inspiration, and there are many who, like myself, owe much to his wise counsel. As a colleague he was always ready with advice, and his wide experience in all branches of medicine made this exceptionally valuable. In addition he had a highly critical appreciation of the legal aspects of his work and was much sought after as a member of various committees. Thomas had a keen sense of humour and an endless supply of anecdotes, but although his hobbies were golf and fishing these subjects were seldom mentioned. He will be greatly missed at Farnborough, especially by those of us who were privileged to enjoy his friendship.

Dr. W. N. GRAY, of Helmsdale, Sutherlandshire, died on January 27, aged 62. William Nicol Gray was born at High Blantyre, Lanarkshire, on August 15, 1894, and was educated at Hamilton Academy. He matriculated at Glasgow University in 1912, his original intention being to become a teacher. His plans were interrupted by the first world war, during which he served first in a munitions factory and then in motor torpedo-boats with the Royal Navy. After the war he turned to medicine and graduated M.B., Ch.B. from Glasgow University in 1922. After holding hospital appointments in Glasgow and Middlesbrough he spent a year as assistant to Dr. W. H. M. Barclay, at Grantown-on-Spey. Though attracted by a country life, he chose to settle in a busy industrial practice in Clydebank in partnership with the late Dr. Thomas M. Strang, a man of outstanding clinical acumen, integrity, and great humanity—traditions which Dr. Gray amply maintained. When the

practice was scattered almost overnight by the fury of the Clydebank "blitz" during the second world war there was widespread dislocation of medical services, and he took up practice again at Helmsdale, where he remained until his death. He leaves a widow and three children, two of whom are graduates in medicine.

R. D. M. writes: In the Highlands outsiders do not always find ready acceptance, but Bill Gray's patent worth, sincerity, depth of understanding, and sense of humour soon made him the friend of all in this scattered area, with its difficult and sometimes snowbound roads. With his passing the Highlands have lost a fine doctor. He identified himself completely with his new parish, even learning Gaelic and later becoming president of the local branch of An Commun Gaidhealach. It would be impossible here to detail the many ways in which Bill Gray served the Highlands during his years in the north. On his shoulders seemed to fall the mantle of that grand old man the late Dr. J. B. Simpson, of Golspie, whom he succeeded as chairman of the Sutherland Insurance Committee. He was also chairman of the Sutherland Division of the British Medical Association and for a year was president of the Northern Counties of Scotland Branch. As a member of the Scottish Committee and the Highlands and Islands Subcommittee of the B.M.A., chairman of the Sutherland Executive Council, of the local medical committee, and of the Highlands and Islands Subcommittee of the Scottish Health Services Council, he undertook a vast amount of extra work and responsibility, as well as many wearisome hours of travel by car and train. Apart from these journeys, he rarely left his practice area. He was a keen fisherman, photographer, and gardener who could coax life out of stones. To his patients, however, he will perhaps always be associated with the struggle to establish the "Hospice" as a small maternity hospital, serving not only his own practice but the rest of the county, particularly the more remote and inaccessible parts. An outstanding all-round clinician with a natural bent for midwifery, he was, moreover, a born teacher; no less than four of his trainees took the D.Obst.R.C.O.G. from this remote country practice. It was a measure of his qualities as a doctor, teacher, and friend that three of his former trainees made the long journey north to join with the sorrowing throng which seemed to include every man in the neighbourhood, who packed the church and streets of Helmsdale to overflowing to bid him sad farewell. In the age of specialization Bill Gray was that fine thing a first-class general practitioner.

Dr. G. W. LLOYD, formerly medical superintendent of the Mayday Hospital, Croydon, and subsequently a general practitioner at Thornton Heath, Surrey, died at the Mayday Hospital on January 28. He was 74 years of age. George William Lloyd was born at Brough, East Yorkshire, on August 1, 1882, and was educated at Hull Grammar School and at St. Bartholomew's Hospital, qualifying M.R.C.S., L.R.C.P. in 1905. After holding a resident appointment at the East Suffolk and Ipswich Hospital he went into a country practice at Alsager, in Cheshire, and while there obtained the London degrees of M.B., B.S. in 1911. He proceeded to the M.D. in 1917. During the first world war he served in the R.A.M.C. from 1914 until he was invalided out of the Army in 1917, and for the next few years was assistant medical superintendent of St. Giles Hospital, Camberwell. From 1922 to 1929 he was medical superintendent of the Mayday Hospital, Croydon. He returned to general practice in 1929, this time at Thornton Heath, and continued in this work until he retired in 1954, a year or two after he had had a stroke. As a young man Dr. Lloyd was a good athlete. He played Rugby for the first fifteen at Bart's and represented the hospital at boxing. In later life his interests were shooting, fishing, ornithology, and the rearing of gun-dogs. He married Miss Elsie Rigby-Jones, of Liverpool, in 1909, and had five sons and one daughter. His eldest son and his son-in-law are both doctors.

Dr. T. M. BODY, who was in practice at Middlesbrough for nearly fifty years, died at Marton-in-Cleveland, Yorkshire, on January 28 at the age of 80. Thomas Munn Body was born at Wittersham, Kent, on December 13, 1876, the son of John Body, J.P., and was educated at Tonbridge School and at St. Bartholomew's Hospital. After qualifying M.R.C.S., L.R.C.P. in 1902 he held the appointments of house-surgeon at the Norfolk and Norwich Hospital and the Royal Portsmouth Hospital and accompanied the Anglo-Liberian Boundary Commission as medical officer. He settled in general practice at Middlesbrough about 1908, and shortly afterwards went into partnership with Dr. John Hedley, succeeding to the practice on Dr. Hedley's death. During the first world war he served as a captain in the R.A.M.C. in France, Mesopotamia, and India. Returning to his practice after the war, he became a prominent figure in the public life of Middlesbrough. He became deputy lieutenant of the North Riding of Yorkshire in 1947, an appointment he regarded not only as an honour to himself but also as one for the medical profession in general. Since 1918 he had been a Justice of the Peace for Middlesbrough, and throughout the second world war he was chairman of the North Riding Territorial Association. He had been chairman also of the committee for the care of mental defectives and of a medical recruitment board at Middlesbrough and president of the Betterment Association. A member of the British Medical Association for forty-five years, he was chairman of the Cleveland Division in 1925–6.

Dr. G. O. MONTGOMERY, who was in general practice at Croydon, Surrey, for some 30 years, died at his home there on January 31 at the age of 63. Geoffrey Owen Montgomery was born in Tasmania on August 31, 1893. He enrolled as a student at University College, London, in 1914, but joined the Army on the outbreak of the first world war. Commissioned in the Green Howards in 1915, he served in France for four years. On return to civilian life he resumed his medical training, and qualified by taking the London Conjoint diploma in 1923. Two years later he obtained the London degrees of M.B., B.S. After holding the resident posts of house-surgeon, house-physician, and obstetric assistant at University College Hospital he became resident medical officer there. In 1927 he joined a firm of doctors at Croydon, and worked in that practice until the time of his death. Among the appointments he held were those of physician to the Croydon General Hospital and medical officer to the Post Office. A keen Territorial officer, he was in France with the British Expeditionary Force in 1940 as commanding officer of the 133rd Field Ambulance, with the rank of lieutenant-colonel. In 1941 he was promoted colonel, and, after serving as assistant director of medical services in the Anti-Aircraft Command, was given the command of a general hospital, first in West Africa and afterwards in India. For his services to the Territorial movement he was awarded the Territorial Decoration in 1955. His cheerful, kindly, and unselfish nature will always be remembered by all who came into contact with him, and a large circle of colleagues, friends, and patients mourn his passing. He leaves a widow and one son and one daughter, to whom our sincere sympathy goes out in their great loss.—E. M. C.

The obituary of Mr. C. L. GIMBLETT was printed in the *Journal* of February 2 (p. 288).

Dr. A. G. SALAMAN writes from Saffron Walden, Essex: When Charles Gimblett came here during the war under the E.M.S. scheme we in these parts met a man of great charm. A complete disregard for pomp was one of his outstanding characteristics. He had an untiring enthusiasm from which he seemed to derive enormous fun even when doing the simplest eye test, and his eagerness to guide us G.P.s through the more treacherous mazes of ophthalmology was a delight to experience. I well remember an occasion when my wife took our son, aged 5, to see him. Gimblett sat solemnly at his desk and asked for symptoms. My son said, "I can't

hardly see nothing at all." With great seriousness Gimblett wrote on the case card : " Patient says, 'I can't hardly see nothing at all.'" He then summoned the patient over in order to sign the statement, which he did in large spidery letters. His vision proved to be normal. Those of us who knew Gimblett remember him as a very human person who, when practising his specialty, never forgot that the owner of the eyes he was examining was a human being too.

Medical Notes in Parliament

DIMINISHED RESPONSIBILITY IN MURDER CASES

The Homicide Bill, emerging without alteration from the committee stage, was given a third reading in the House of Commons on February 6—the voting was 217 to 131—and sent up to the House of Lords.

Mr. R. A. BUTLER moved the third reading in his first speech as Home Secretary, and in so doing sought to put the Government's proposals in a new setting. The Bill represented some progress, he said, since it swept away the doctrine of constructive malice and enlarged the defence of provocation ; but, more particularly, in its adaptation to English law on the doctrine of diminished responsibility it would enable the courts to take account of the great strides that had been made in the understanding of the human mind, and to avoid treating as murder killings for which the accused ought not to be held wholly responsible. This, he went on, was all part of the new approach to the problems of the mind which would exercise both the Home Office and Parliament in the future. This unexplored territory of the mind would be perhaps one of the most exciting of the adventures before the human intellect ; it was as important in the human sphere as nuclear development was in the scientific. He invited in anticipation the support of the House in any further moves in the direction of the humanizing and understanding of relationships.

This part of his speech at any rate was welcomed by the Opposition, though their general opinion of the Bill was that for the sake of party unity the Government had missed the opportunity of what could have been a great measure of penal reform. Mr. SYDNEY SILVERMAN (Nelson and Colne, Lab.) moved the official amendment against the third reading which, while welcoming the removal of some anomalies in the law of murder and the abolition of the death penalty from some offences, refused assent to a Bill which " fails to satisfy the public sense of justice, and is in defiance of the principle which the House has already approved—namely, that the death penalty for murder no longer accords with the needs or the true interests of a civilized society."

Indefinite Sentences

Dr. REGINALD BENNETT (Gosport and Fareham, Con.) said he felt considerable unease of mind about Clause 2. A verdict reduced to manslaughter automatically on proof of diminished responsibility meant the introduction of a new code on which the courts would have to decide these matters which would exist simultaneously—and in conflict—with the McNaughton Rules. Which of these two mechanisms was to be invoked ? If there was prima facie abnormality of mind, did a charge of murder still stand ? If it was evident from unopposed medical evidence before a trial that the prisoner's mental state was in other ways deranged, did the indictment drop to manslaughter ? The greater difficulty was of deciding on what basis and to what degree there was irresponsibility. The provision in Clause 2 was whether the accused " was suffering from such abnormality of mind (whether arising from a condition of arrested or retarded development of mind or any inherent causes or induced by disease or injury) as substantially impaired his mental responsibility for his acts or omissions." The McNaughton Rules test was whether the accused was suffering at the time from such a defect of mind as to be unable to appreciate the nature of his act or the fact that it was wrong. Which standard came first ? Which was to be a criterion ? If a person was found guilty of manslaughter there could be little purpose in sending him to a psychopathic institution, with a finite sentence.

The East-Herbert institution being built was not likely to be effective or to give anything like the results that the Herstedvester Institution in Denmark was giving, unless they were prepared to agree that for curative purposes the mentally afflicted offender must be sent to such an institution for an indefinite sentence, with the varying degrees of probation that would follow it as his rehabilitation became more complete. If the McNaughton Rules applied, a man found to be suffering from a defect of reason would go to Broadmoor, which was automatically an indefinite sentence. If, however, his defect of mind—maybe even the same defect —was found to justify a sentence for manslaughter, he would go down for seven years, and there would be no consideration of any treatment for his mental state except such as was incidental to an ordinary term of imprisonment.

Mr. J. E. S. SIMON, Under-Secretary, Home Office, saw the introduction of the defence of diminished responsibility as possibly the greatest change that was being made in the law of England. Among other factors, it provided a criterion which took cognizance of the fact that there were many degrees of mental responsibility for crime. The relation between the two defences, of insanity and diminished responsibility, he suggested was this. The accused man might, on the face of it, be unfit to plead. That was generally determined initially by the prosecution, although it might be raised by the defence, and a jury was empanelled to decide whether the man was fit or unfit to plead. Only if the man was fit to plead did any question of these defences arise, and then it was a matter for the man: it was for him to say whether he would raise a defence which would lead to a verdict of " Guilty but insane," or a defence which would lead to a verdict of manslaughter by raising a question of diminished responsibility. The one was under the McNaughton Rules ; it was a complete defence, and the verdict was " Guilty but insane," equivalent to " Not guilty." The diminished responsibility defence would lead to a verdict of " Guilty," but " Guilty of manslaughter." It did not necessarily follow that the man would be sentenced to a fixed term. It was entirely a matter for the judge's discretion, on the evidence given to him after the verdict, whether he passed a sentence for life if he could not determine the time necessary to treat the prisoner successfully. He could sentence the convicted man to a term of years. Equally he could, if he wished, put him on probation for the crime of manslaughter, and make it a condition in a proper case that the man should submit himself to psychiatric treatment, or any other medical treatment, for a suitable time.

Poliomyelitis Vaccination

Mr. M. TURNER-SAMUELS (Gloucester, Lab.) asked the Minister of Health on February 8 whether, in view of the fact that the remuneration to general practitioners for carrying out the responsible duties and complicated arrangements required to collect, store, and administer the vaccine and keep the necessary records in connexion with inoculation against poliomyelitis was unreasonably low, he would increase the present inclusive fee of 5s. for the two inoculations required in each case to a more reasonable figure. Mr. D. VOSPER stated that the vaccination would be performed as part of the general practitioner's comprehensive medical care of his patients for which he received remuneration from his local executive council. The additional 5s. fee was to be paid by the local health authority for the record of vaccination. This was the present fee for records of other forms of immunization, and was agreed between representatives of local authorities and of the medical profession.

Analgesia

Mr. R. W. SORENSEN (Leyton, Lab.) asked what further advance had been made in the use of analgesia and the supply of apparatus for this purpose in hospitals and in domiciliary practice; and, in the light of experience during the past year, what was the merit and cost of trilene, as compared with analgesia, in respect of confinements. Mr. VOSPER replied that no information was available about the use of analgesics in hospitals or by general practitioners. In 1955 inhalational analgesics were administered by domiciliary midwives in 75% of the confinements attended by them, compared with 72% in 1954, and the number of sets of apparatus in use rose from 6,489 to 6,764. He was advised that trilene served broadly the same clinical purposes as gas and air, but the apparatus was more easily transportable. The costs of the types of apparatus in general use were broadly comparable. The use of trilene, he stated, was on the increase. At the end of 1955 about 256 trilene sets were in use, and this had risen at the end of 1956 to about 900.

Health Centres

Mr. J. K. VAUGHAN-MORGAN, Parliamentary Secretary, Ministry of Health, informed Mr. W. T. PROCTOR (Eccles, Lab.) on February 4 that eight new health centres had been provided under the Health Service since July, 1948. In addition, premises were taken over in July, 1948, by six local authorities so that general medical or dental services could be continued there. Current restrictions on capital expenditure rule out further provision for the time being. The new centres were: Faringdon, Berks; William Budd, Knowle West, Bristol; Woodberry Down, Stoke Newington, London; John Ryle Health Centre, Clifton Estate, Nottingham; Harold Hill, Romford Aveley, Thurrock, Essex; Hesters Way Estate, Cheltenham; and Alderman Jack Cohen Health Centre, Sunderland.

Pay Variations

Dr. DONALD JOHNSON (Carlisle, Con.) asked the Minister whether he was aware that the average earnings of general practitioners supplied by his department was comprised of wide variations; and whether he would give a breakdown of such an average. Mr. VOSPER, in reply, said that the breakdown of earnings for which he asked was not available.

Mr. J. A. LANGFORD-HOLT (Shrewsbury, Con.) asked what was the average salary received by doctors working whole-time for the Health Service after deduction of income tax. Mr. VOSPER told him also that this information was not available.

Dental Council's Rebuff

Mr. BARNETT JANNER (Leicester, North-west, Lab.) asked for a statement on the availability of dental hygienists for the general public in accordance with the provisions of the Dentists Act, 1956.

Mr. VOSPER replied that some dental hygienists were already employed in the local authority and hospital dental services, but they could not work with dentists in private practice until permitted by regulations made under the Dentists Act, 1956, by the General Dental Council. The General Dental Council had asked the Privy Council to approve draft regulations which provided that dental hygienists should not be allowed to work in private practice until such time as the requirements of the local authority and hospital dental services were in the opinion of the General Dental Council satisfied. This limitation would not appear to accord with the intentions of Parliament, as expressed during the passage of the Bill, and the proposals had therefore been referred back to the General Dental Council for further consideration.

INFECTIOUS DISEASES AND VITAL STATISTICS

Summary for British Isles for week ending **January 26** (No. 4) and corresponding week 1956.

Figures of *cases* are for the countries shown and London administrative county. Figures of *deaths* and *births* are for the 160 great towns in England and Wales (London included), London administrative county, the 17 principal towns in Scotland, the 10 principal towns in Northern Ireland, and the 14 principal towns in Eire.

A blank space denotes disease not notifiable or no return available.

The table is based on information supplied by the Registrars-General of England and Wales, Scotland, N. Ireland, and Eire, the Ministry of Health and Local Government of N. Ireland, and the Department of Health of Eire.

CASES in Countries and London	1957					1956				
	Eng. & Wales	Lond.	Scot.	N. Ire.	Eire	Eng. & Wales	Lond.	Scot.	N. Ire.	Eire
Diphtheria	1	0	5	0	1	4	1	7	0	5
Dysentery	635	43	255	0	1	867	94	175	11	2
Encephalitis, acute	5	0		0		5	0		1	
Enteric fever:										
Typhoid	1	0	0	0		2	0	0	0	
Paratyphoid	2	0	1(B)	0		4	0	1(B)	1	
Food-poisoning	165	20	46	5		92	17		0	
Infective enteritis or diarrhoea under 2 years	.			14	16				10	15
Measles *	9,527	682	431	428	201	2,130	22	85	21	108
Meningococcal infection	23	2	17	0	1	31	1	13	2	1
Ophthalmia neonatorum	27	0	4	0		35	2	6	0	
Pneumonia †	572	35	338	21		836	60	406	17	2
Poliomyelitis, acute:										
Paralytic	21	2	2	0	11	21	1	2	3	3
Non-paralytic	6	0				10	2			
Puerperal fever §	241	28	9	1	1	263	38	4	0	
Scarlet fever	829	47	98	36	20	964	52	110	41	27
Tuberculosis:										
Respiratory	636	70	116	26		700	94	123	21	
Non-respiratory	30	4	9	2		102	5	8	8	
Whooping-cough	3,233	154	506	14	65	1,537	90	88	108	224

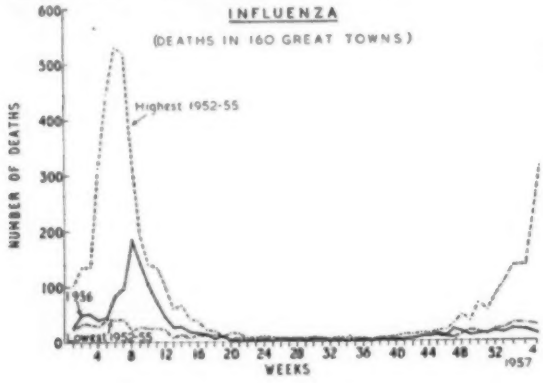
DEATHS in Great Towns	1957					1956				
	Eng. & Wales	Lond.	Scot.	N. Ire	Eire	Eng. & Wales	Lond.	Scot.	N. Ire.	Eire
Diphtheria	0	0	0	0	1	0	0	0	0	0
Dysentery	1	0		0		0	0		0	
Encephalitis, acute		0			0		0			0
Enteric fever	0	0	0	0		0	0	0	0	
Infective enteritis or diarrhoea under 2 years	8	0	0	1	2	7	0	1	0	0
Influenza	12	0	3	1	1	39	7	6	0	1
Measles		0	0	0	0		0	0	0	0
Meningococcal infection		1	0				0	0		
Pneumonia	282	46	32	17	13	373	72	46	19	7
Poliomyelitis, acute	2	2			0	2	1			1
Scarlet fever		0	0	0	0		1	0	0	0
Tuberculosis:										
Respiratory	52	3	6	2	1	72	13	19	2	3
Non-respiratory		0	2	0	1		0	0	0	0
Whooping-cough	0	0	1	0	0	0	0	0	1	1
Deaths 0–1 year	220	21	32	7	26	249	33	30	11	9
Deaths (excluding stillbirths)	5,944	784	712	156	190	6,735	1030	795	134	181
LIVE BIRTHS	7,982	1125	1000	222	331	7,719	1155	884	223	369
STILLBIRTHS	199	18	21			219	36	19		

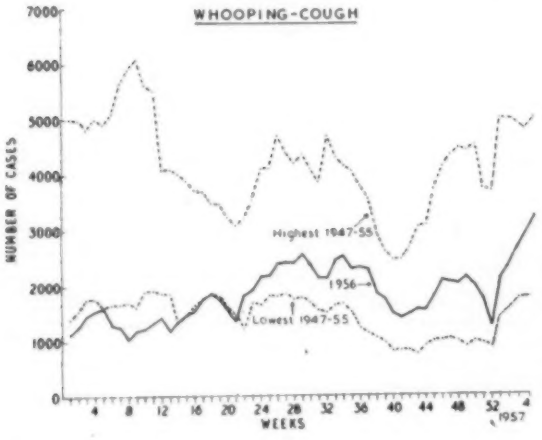
* Measles not notifiable in Scotland, whence returns are approximate.
† Includes primary and influenzal pneumonia.
§ Includes puerperal pyrexia.

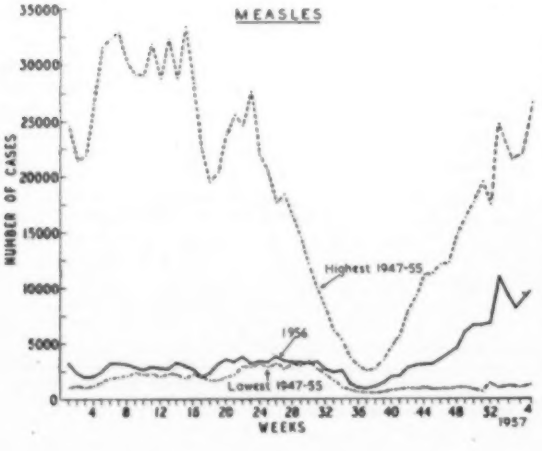
Vital Statistics

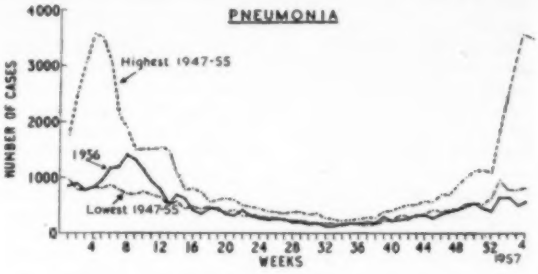
Graphs of Infectious Diseases

The graphs below show the uncorrected numbers of cases (deaths for influenza) of certain diseases notified weekly in England and Wales (great towns for influenza). Highest and lowest figures reported in each week during the years 1947–55 (influenza, 1952–5) are shown thus ------, the figures for 1956–7 thus ———. Except for the curves showing notifications in 1956–7, the graphs were prepared at the Department of Medical Statistics and Epidemiology, London School of Hygiene and Tropical Medicine.









Infectious Diseases

Infectious diseases were more prevalent in England and Wales during the week ending January 26. Rises in the numbers of notifications included 1,352 for measles, from 8,175 to 9,527, 569 for whooping-cough, from 2,664 to 3,233, 158 for dysentery, from 477 to 635, 136 for scarlet fever, from 693 to 829, and 56 for acute pneumonia, from 516 to 572.

The largest rises in the incidence of measles were 373 in Lancashire, from 1,931 to 2,304, 202 in Lincolnshire, from 62 to 264, 174 in Northumberland, from 472 to 646, 166 in Surrey, from 238 to 404, 144 in Durham, from 666 to 810, 130 in Essex, from 673 to 803, and 107 in Cumberland, from 142 to 249; the chief exceptions to the general trend were falls of 126 in Yorkshire West Riding, from 610 to 484, and 72 in Oxfordshire, from 159 to 87. The largest rises in the number of notifications of whooping-cough were 63 in Suffolk, from 36 to 99, 47 in Lancashire, from 297 to 344, and 40 in Kent, from 168 to 208. The largest increases in scarlet fever were 45 in Shropshire, from 2 to 47, due to an outbreak involving 44 persons in Bridgnorth R.D., and 35 in Essex, from 30 to 65. Only one case of diphtheria was reported, 3 fewer than in the preceding week.

27 cases of acute poliomyelitis were notified, and these were 9 fewer for paralytic and 5 fewer for non-paralytic cases than in the preceding week. The largest returns were 3 cases in the counties of Lancashire, Essex, and Hertfordshire (Stevenage U.D. 2).

The rise in the incidence of dysentery was due to an increase in established outbreaks rather than to new centres of infection. The largest returns were Lancashire 121 (Stretford M.B. 49, Liverpool C.B. 13), Warwickshire 89 (Coventry C.B. 47, Birmingham C.B. 20), Yorkshire West Riding 80 (Bradford C.B. 14, Sheffield C.B. 14, Leeds C.B. 12, Keighley M.B. 11); Yorkshire East Riding 45 (Kingston-upon-Hull C.B. 42), London 40 (these cases were scattered through 15 boroughs), Nottinghamshire 35 (Arnold U.D. 18, Nottingham C.B. 15), Northumberland 29 (Alnwick U.D. 13), Staffordshire 22 (West Bromwich C.B. 13), Surrey 21.

Week Ending February 2

The notifications of infectious diseases in England and Wales during the week included: scarlet fever 865, whooping-cough 3,302, diphtheria 5, measles 13,008, acute pneumonia 661, acute poliomyelitis 37, dysentery 1,097, paratyphoid fever 10, and typhoid fever 3.

Isle of Man Census

The total population enumerated in the Isle of Man at the 1951 census was 55,253, representing a net gain since 1931 of about 6,000 persons. 1,229 visitors were enumerated in the Isle of Man, and 1,306 persons enumerated in the United Kingdom stated that their usual place of residence was in the Isle of Man. It is estimated that 55,437 persons were usually resident in the Isle of Man at the census date.

Householders were asked to state whether they had exclusive use of, shared with another household, or were without piped water supply within the house, cooking stove or range, kitchen sink, water-closet, or fixed bath. 55% of all private households had exclusive use of all five of these

household arrangements, and a further 14% had all except a fixed bath; these figures compare with 52 and 19% for England and Wales.

The population of the island was on the whole a little older than that of England and Wales. 13.9% were aged 65 and over, compared with 10.9% in 1931. The figures for England and Wales were 11.0 in 1951 and 7.4 in 1931. The ratio of females to males in the Isle of Man was higher than in England and Wales, the numbers of females per 1,000 males being respectively 1,144 and 1,082. A striking feature of the returns was the increase since 1931 in the proportions married, particularly among younger people. At age 20–24 there were 169 males married per thousand as compared with only 96 in 1931, and 401 females per thousand compared with 169.

Total fertility in the Isle of Man was somewhat higher than in England and Wales. There were 173 live-born children per 100 married women under age 50, compared with 169 in England and Wales. Current fertility rates (percentages of married women under age 50 bearing a live-born child in the year ending April 8, 1951) were slightly higher in the Isle of Man (10%) than in England and Wales (9%).

The number of people returned in 1951 as able to speak Manx (all of whom could speak English also) was 355 for the whole island, compared with 4,657 in 1901, 915 in 1921, and 529 in 1931.

Medical News

Radiological Practice.—The Minister of Health and the Secretary of State for Scotland have now appointed the promised committee under Lord ADRIAN'S chairmanship to review current practice in diagnostic radiology and the radiotherapy of non-malignant conditions, and it has started its work. The committee's appointment was recommended by the Medical Research Council subcommittee responsible for the preparation of the Council's report on *The Hazards to Man of Nuclear and Allied Radiations* (Cmd. 9780, H.M.S.O., 1956). Besides reviewing current practice in the two fields of radiology mentioned, the committee is asked to submit recommendations to the Ministers. The members of the committee in addition to Lord Adrian are: Professor J. C. McC. Browne (professor of obstetrics and gynaecology, London), Sir Horace Evans (director, medical unit, London Hospital), Mr. R. J. G. Grewcock (dental surgeon, London), Professor R. J. Kellar (professor of obstetrics and gynaecology, Edinburgh), Dr. Peter Kerley (director, *x*-ray unit, Westminster Hospital), Dr. D. R. Maitland (radiologist, Royal Infirmary, Edinburgh), Professor W. V. Mayneord (professor of medical physics, London), Professor J. S. Mitchell (professor of radiotherapeutics, Cambridge), Mr. C. Naunton Morgan (surgeon, St. Bartholomew's Hospital), Dr. Kenneth Robson (chest physician, St. George's Hospital), Mr. T. Holmes Sellors (thoracic surgeon, Middlesex Hospital), Mr. G. R. Seward (dental surgeon, Ilford), Professor F. W. Spiers (professor of physics, Leeds), Professor E. J. Wayne (regius professor of the practice of medicine, Glasgow), Professor Charles A. Wells (professor of surgery, Liverpool), and Professor B. W. Windeyer (professor of radiotherapy, London).

Research on Smoking and Lung Cancer.—Following a question in Parliament this month about public expenditure on research on smoking and lung cancer (*Journal*, February 9, p. 350), the Tobacco Manufacturers' Standing Committee has released, by agreement with the Medical Research Council, some details from the latter's report on the projects supported from the Tobacco Manufacturers' research fund. The report covers the year which ended on October 31 last. During that period the total expenditure from the fund was £51,679, of which £28,679 was non-recurrent and £23,000 recurrent. This brings the total so far spent from the £250,000 fund to £68,679. (The fund was accepted by the M.R.C. three years ago.) The major grants made from the fund during 1956 were for the expansion of the work at St. Bartholomew's Hospital on atmospheric pollution, and for the establishment of a group at Exeter to study the chemistry of tobacco smoke. In addition, states the report, "there are other important projects which have only just got under way." In America the Tobacco Industry Research Committee there announces that it made grants to the value of $566,000 during 1956, bringing the total sum allocated since the latter part of 1954 to $1,380,000.

World Federation for Mental Health.—The executive board of the World Federation for Mental Health has been meeting in London, and on February 7 the Federation's United Kingdom committee held a press reception in honour of Dr. MARGARET MEAD, this year's president of the Federation. She was introduced by Dr. J. R. REES, director of the Federation. Earlier, on January 30, Dr. Mead had delivered the British Psycho-Analytical Society's eighth Ernest Jones lecture at the Royal Society of Medicine, and on February 8 she gave a public lecture on "Mental Health in a Changing World" at County Hall. Dr. Mead, whose work on primitive cultures is well known from her books, is now particularly concerned with the impact of social forms and cultural changes on the development of character. She is associate curator of ethnology at the American Museum of Natural History in New York. Among those who attended the reception were Dr. BROCK CHISHOLM, of Canada, W.H.O.'s first director-general, Dr. F. FREMONT-SMITH, medical director of the Josiah Macy, Jr., Foundation, New York, and Dr. EDUARDO KRAPF, of Buenos Aires, chief of W.H.O.'s mental health section.

Polish Surgeons Visit Britain.—Two Polish surgeons, Dr. WITOLD RUDOWSKI, chief surgeon at the Radium Institute, Warsaw, and Dr. BRONISLAW PRASZALOWICZ, senior surgeon at the Institute of Haematology, Warsaw, and deputy editor-in-chief of the Polish State Medical Publishing House, have been visiting Britain under British Council auspices. They arrived on February 2 and have been seeing hospitals and university departments in London, Manchester, Edinburgh, Oxford, and Birmingham. With them has been Mr. TADEUSZ KLONOWICZ, director of the Polish State Medical Publishing House. Their visit, which is a return of that paid to Warsaw last autumn by Sir ROBERT MACINTOSH, Professor IAN AIRD, and Dr. R. H. DOBBS (see *Journal*, November 24, p. 1230), ends to-day.

Tata Awards for Leukaemia Research.—The trustees of the Lady Tata Memorial Fund invite applications for grants, scholarships, or fellowships for research on diseases of the blood, with special reference to leukaemia, in the academic year beginning on October 1, 1957. Candidates with programmes of research on any aspect of malignant disease which may throw incidental light on leukaemia will be eligible for consideration. The awards are open to suitably qualified investigators of any nationality, working in any country in which it will be possible to make payments.

Grants of variable amount are made for research expenses or to provide scientific or technical assistants for senior workers. Scholarships are awarded for the personal remuneration of workers carrying out approved research under suitable direction; their normal value will be £600 sterling per annum for whole-time research, with proportional adjustment for work on a part-time basis. In addition, the trustees may award one or more Lady Tata Memorial Research Fellowships to workers with considerable research experience; the stipends of these fellowships will be at the rate of £1,200 sterling per annum.

Further particulars and forms of application may be obtained from the Secretary of the (European) Scientific Advisory Committee, Lady Tata Memorial Trust, Chester Beatty Research Institute, Fulham Road, London, S.W.3. Applications must be submitted before March 31, and the awards will be announced in June.

French Government Exchange Scholarships.—Under the terms of a reciprocal arrangement with the Centre National

de la Recherche Scientifique, the Medical Research Council invites applications from British workers in medical science for two scholarships tenable at research institutions in France during the academic year 1957–8. The scheme is intended for young graduates in medicine or science who have already had some experience in a branch of medical science. Candidates must be of British nationality and have at least a working knowledge of French.

The scholarships will normally carry a maintenance allowance within the range of 50,000–70,000 francs a month, according to qualifications and experience, and married men will receive a supplementary allowance (payable in sterling by the M.R.C.). Provision will also be made for travelling expenses to and from the place of work.

Application forms may be obtained from the Secretary, Medical Research Council, 38, Old Queen Street, London, S.W.1, with whom applications must be lodged by March 31.

Polio Vaccination at Wakefield and Burton-on-Trent.—Neither Wakefield nor Burton-on-Trent participated in the Ministry of Health's poliomyelitis vaccination programme last year, as a result of advice given by their health committees. This year they are participating. On December 4 the Wakefield City Council resolved to give children in their area, born between 1947 and 1954, an opportunity to be vaccinated against poliomyelitis. The Burton Borough Council took its decision last April, but it was then too late to register the children for the 1956 vaccination programme.

International Society of Internal Medicine.—Sir RUSSELL BRAIN, P.R.C.P., has been elected president of the International Society of Internal Medicine. He took office on February 1. The Society's next meeting will be held in Philadelphia from April 24 to 26, 1958.

Eli Lilly Fellowship (South Africa).—Dr. H. A. SHAPIRO, as honorary chairman of the selection committee, writes to draw the attention of medical practitioners registered in South Africa but resident at present abroad to the 1957 Eli Lilly medical research fellowship. This fellowship is for research in the United States and provides a net sum of 250 dollars a month together with travelling expenses to and from the place of study. The closing date for applications is April 30. Further details may be obtained from Dr. Shapiro, P.O. Box 1010, Johannesburg.

COMING EVENTS

William Sheen Memorial Lecture.—Sir HARRY PLATT, P.R.C.S., will deliver the Welsh National School of Medicine's sixth William Sheen Memorial lecture on March 6 at 8 p.m. in the Reardon-Smith lecture theatre, Park Place, Cardiff. His subject will be "The Backache–Sciatica Problem."

Royal Society of Health.—Exhibition on "Atoms and Health" at 90, Buckingham Palace Road, London, S.W.1, March 18 to May 31, Mondays to Fridays, 10 a.m. to 5 p.m.

Physical Society.—Exhibition of scientific instruments and apparatus at the Royal Horticultural Society's Old and New Halls, Westminster, London, S.W.1, March 25–28. Tickets from the secretary-editor, 1, Lowther Gardens, London, S.W.7.

SOCIETIES AND LECTURES

A fee is charged or a ticket is required for attending lectures marked ●. Application should be made first to the institution concerned.

Saturday, February 16

KENT PAEDIATRIC AND CHILD HEALTH SOCIETY.—At Children's Department, Farnborough Hospital, 3 p.m., Dr. J. D. Kershaw: Child Health in the U.S.S.R. (illustrated by slides).

Monday, February 18

HUNTERIAN SOCIETY.—At the Mansion House, 8.30 p.m., Hunterian Oration by Mr. A. W. Badenoch: The Surgery that Hunter Changed.

POSTGRADUATE MEDICAL SCHOOL OF LONDON.—4 p.m., Dr. W. Ritchie Russell: Pain

●UNITED HOSPITALS CATHOLIC SOCIETY.—At 13, Devonshire Place, W., 6.15 p.m., Mr. John Ryan: Family Limitation.

UNIVERSITY COLLEGE LONDON: DEPARTMENT OF PHARMACOLOGY.—At Physiology Theatre, Gower Street, W.C., 5.30 p.m., Dr. T. S. Work: Biochemical Approach to the Problems of Protein Synthesis.

Tuesday, February 19

BRITISH POSTGRADUATE MEDICAL FEDERATION.—At London School of Hygiene and Tropical Medicine, 5.30 p.m., Dr. A. L. Greenbaum, Ph.D.: Control of Fat Metabolism.

ILFORD MEDICAL SOCIETY.—At King George Hospital, 8.45 p.m., Dr. James Graham: Planning of Hospital Services on a Regional Basis. Members of Stratford Division, B.M.A., are invited.

INSTITUTE OF DERMATOLOGY.—5.30 p.m., Dr. G. B. Dowling: Scleroderma and Dermatomyositis.

ROYAL ARMY MEDICAL COLLEGE.—5 p.m., Professor G. W. A. Dick: Attenuated Virus Vaccines.

ROYAL COLLEGE OF SURGEONS OF ENGLAND.—3.45 p.m., Arnott Demonstration by Dr. Frank Stansfield: Anatomy of the Visual Pathway.

ST. MARY'S HOSPITAL MEDICAL SCHOOL.—At Wright-Fleming Institute Theatre, 5 p.m., Dr. N. F. Morris: Uterine Blood Flow During Late Pregnancy and Labour.

SOUTH-WEST LONDON MEDICAL SOCIETY.—At Bolingbroke Hospital, Wandsworth Common, S.W., 8.30 p.m., Mr. R. W. Nevin: Ulcerative Colitis.

Wednesday, February 20

INSTITUTE OF DERMATOLOGY.—5.30 p.m., Dr. H. Haber: Localized Disease of the Collagen.

INSTITUTE OF DISEASES OF THE CHEST.—5 p.m., Dr. H. Nicholson: Corticosteroids in Chest Disease.

INSTITUTE OF UROLOGY.—4.30 for 5 p.m., Mr. A. Clifford Morson: Non-tuberculous Infections of the Urinary Tract.

POSTGRADUATE MEDICAL SCHOOL OF LONDON.—2 p.m., Dr. J. H. Humphrey: Biochemical Mechanisms Underlying Hypersensitivity Reactions (D.

ROYAL MEDICAL SOCIETY, EDINBURGH.—2.30 p.m., Industrial Visit to Caledonian Distillery, Edinburgh.

Thursday, February 21

AUSTRIAN INSTITUTE, 28, Rutland Gate, S.W.—8.30 p.m., Professor V. Frankl (Vienna): Collective Neuroses of the Present Day.

BRITISH POSTGRADUATE MEDICAL FEDERATION.—At London School of Hygiene and Tropical Medicine, 5.30 p.m., Dr. Honor B. Fell, Ph.D., D.Sc., F.R.S.: Physiology of Skeletal Tissue in Culture.

EDINBURGH CLINICAL CLUB.—At Royal College of Physicians of Edinburgh, 8 p.m., Dr. G. Swapp: Organization in General Practice.

GLASGOW UNIVERSITY MEDICO-CHIRURGICAL SOCIETY.—7.30 p.m., Mr. J. Dunbar: Personalities in Medicine.

HILL END HOSPITAL MEDICAL SOCIETY, St. Albans.—8.45 p.m., Dr. W. Edwards: Changing Picture of Tuberculosis.

HYPNOTHERAPY GROUP.—At Royal Society of Medicine, 8 p.m., Dr. D. M. L. Doran and Dr. I. Hadfield: Hypnotherapy from the Trainee Point of View. A discussion will follow.

LIVERPOOL MEDICAL INSTITUTION.—8 p.m., pathological meeting. Mrs. Edith K. Dawson, M.D.: Mammary Cancer in Young Subjects, with Special Reference to Pregnancy.

LONDON JEWISH HOSPITAL MEDICAL SOCIETY.—At Medical Society of London, 8.30 p.m., Professor C. G. Rob: Surgery of Arterial Disease.

NORTH-EAST CLINICAL SOCIETY.—At Prince of Wales's General Hospital, N., 2.30 p.m., clinical meeting.

NORTH-WESTERN TUBERCULOSIS SOCIETY.—At Clinical Theatre, Manchester Royal Infirmary, 5 p.m., Dr. W. Brockbank: Some Aspects of Asthma.

ROYAL COLLEGE OF SURGEONS OF ENGLAND.—3.45 p.m., Arnott Demonstration by Mr. B. E. Greenfield: Functions of the Masseter Muscle. Studies on Normal and Abnormal Subjects.

ROYAL SOCIETY OF TROPICAL MEDICINE AND HYGIENE.—At 26, Portland Place, W., 7.30 p.m., Dr. W. S. S. Ladell: Some Physiological Aspects of the Heart Disorders. A discussion will follow.

ST. GEORGE'S HOSPITAL MEDICAL SCHOOL.—5 p.m., Dr. D. J. Williams: neurology demonstration.

WEST OF ENGLAND CHILD HEALTH GROUP.—At Department of Child Health Lecture Theatre, Bristol Royal Hospital for Sick Children, 5.30 p.m., Dr. J. C. Spencer, Ph.D.: Neighbourhood and Family.

Friday, February 22

●INSTITUTE OF DERMATOLOGY.—5.30 p.m., Dr. A. D. Porter: clinical demonstration.

INSTITUTE OF DISEASES OF THE CHEST.—5 p.m., Dr. K. Robson: clinical demonstration.

POSTGRADUATE MEDICAL SCHOOL OF LONDON.—10 a.m., Mr. G. Flavell: Unusual Manifestations of Cancer of the Lung.

ROYAL MEDICAL SOCIETY, EDINBURGH.—8 p.m., address by Professor A. Kennedy: Interrogation and Brainwashing.

ROYAL MEDICO-CHIRURGICAL SOCIETY OF GLASGOW.—At Royal Faculty of Physicians and Surgeons of Glasgow, 8.30 p.m., reception and dance.

Saturday, February 23

BIOCHEMICAL SOCIETY.—At Beveridge Hall, Senate House, London University, 11 a.m., Symposium: Structure and Function of Subcellular Components.

BIRTHS, MARRIAGES, AND DEATHS

BIRTHS

Evans.—On December 23, 1956, at Morfa, New Park Road, Holyhead, Anglesey, to Dr. Joan Evans (formerly Wilson) and Dr. Emrys Evans, a brother for David.

DEATHS

Crymble.—On January 26, 1957, Edward Marshall Tate Crymble, L.R.C.P. &S.I. and L.M., of 27, Derby Road, Fallowfield, Manchester.

Kay.—On January 15, 1957, at Boscombe Hospital, Charles Wallace Kay, L.R.C.P.&S.Ed., L.R.F.P.S.

Macdowel.—On January 16, 1957, Francis Lewis Hartwell Macdowel, L.R.C.P.&S.I. and L.M., Surgeon Captain, R.N. (retired), of The Skippets, Sheepscombe, near Stroud, Glos.

Newton.—On January 17, 1957, Herbert William Newton, M.R.C.S., L.R.C.P., late of Chester Cottage, Kingsdown, Deal, Kent, formerly of Feltham, Middx, aged 90.

Orchard.—On January 15, 1957, at 458, New Bedford Road, Luton, Beds, Harry Percival Orchard, M.R.C.S., L.R.C.P., late of Brixton, London, S.W., aged 76.

Any Questions?

We publish below a selection of those questions and answers which seem of general interest. It is regretted that it is not possible to supply answers to all questions submitted.

Third Shot of Polio Vaccine

Q.—*(1) Does the present evidence suggest that a third inoculation with a Salk-type vaccine is desirable? (2) If so, when is the optimum time to give it, and how long after the second injection does it still remain worth doing? (3) Does it matter if the vaccine used for the third injection, although of Salk-type, is not identical with that used for the first two inoculations?*

A.—(1) With the Salk-type vaccines at present available there is little doubt that a third inoculation is needed before a person can be considered adequately immunized. This should be given from six to twelve months after the initial course of two injections spaced four to six weeks apart. By this time the antibody level obtained after the first two injections will be reaching a low level in many of the vaccinated, and the third injection will "boost" it, often to that obtained after natural infection. The fall after this "booster" is very much slower than after the initial course, and antibody will probably remain at detectable levels for at least three to four years.

(2) The ability to respond to a "booster" almost certainly persists longer than detectable antibody, and the third dose can act in this way two to three years after the initial course. As it is not known if the secondary reaction to natural infection is quick enough in these circumstances to prevent any virus reaching the central nervous system, it is preferable not to delay the third injection for more than a year, so that some antibody may be maintained in the circulation continuously. In diphtheria it has been shown that, when the level of antitoxin in the serum is very low, clinical disease may occur before the secondary antibody rise has had time to be effective. Of the inoculated subjects 20% will not respond to the first injection, but do so to the second; a third injection is essential in these people, and this is another reason why it should not be delayed for more than a year.

(3) The type of Salk vaccine used is not important, provided it has been produced under regulations ensuring adequate potency and safety, as the polio viruses fall into three distinct antigenic types with little antigenic variation within each.

Hay Fever Season

Q.—*When is the usual hay fever season in Britain, and for how long does it last? To what extent do variations in locality, flora, weather, or other factors influence the time of its onset or its duration? For example, what difference would be expected in the hay fever seasons in Devon and the West Highlands of Scotland?*

A.—Grass pollen, the most important cause of hay fever, is most prevalent in the air in England and Wales during the period June 1 to July 15.[1] The hay fever season opens during the last week of May, is at its height in early and mid-June and continues for a variable period (depending on the individual patient's degree of sensitivity), and ends during the latter part of July. The severity of the grass-pollen season is related to the luxuriance of the season's grass crop, which in its turn depends mainly on the rainfall in April and May[2]; but, since the release of pollen into the air is dependent on bright sunshine, sunny periods are much more conducive to dense grass-pollen clouds—and to hay fever—than are dull ones. During an abnormally sunless, cool, and wet summer only about half as much pollen is released into the air as in a sunny, hot, and dry one. The densest clouds of grass pollen are experienced in the neighbourhood of meadows of standing mowing-grass during the hours of pollen liberation. This period varies from species to species: in most, though not all, species it occurs in the early morning or the late afternoon. In general, the more remote a particular place is from large areas of cultivated grassland, the lower is the grass-pollen incidence.

No figures are available for either Devon or the West Highlands of Scotland, but to judge from the evidence from lowland and mountain areas, respectively, one would expect Devon to be much more subject to grass pollen than the West Highlands. What evidence there is suggests also that the grass-pollen season opens perhaps 10 days later in Scotland than in southern Britain.

It has been found that very significant quantities of pollen of all kinds are carried from productive areas into non-productive land areas and to a distance of at least 30 miles out to sea.

REFERENCES

[1] Hyde, H. A., *British Medical Journal*, 1949, 1, 897.
[2] —— *Acta allerg. (Kbh.)*, 1952, 5, 98.

Varicose Eczema

Q.—*Are local applications of cortisone ointment of value in the conservative treatment of varicose eczema? My patient refuses operation. What alternative treatment is advised?*

A.—True varicose eczema occurs only in the presence of chronic venous insufficiency with oedema. No local measures will improve the eczema unless the oedema is abolished. Rest in bed will accomplish this, but, although occasionally necessary, it is undesirable for social and economic reasons and because of the risk of thrombophlebitis. Elastic compression is best, though elastic stockings are unsuitable.

Once the affected area is dry, elastic crêpe bandages may be used, but if there is much weeping ichthyol paste bandages covered by elastic adhesive bandages are very efficacious. They should be changed weekly during the weeping stage, which is usually soon passed, and less frequently once a dry glazed surface appears. The Unna's paste type of bandage is not well tolerated in the presence of varicose eczema. During the period of absorption of oedema fluid from an eczematous leg, patches of eczema may appear on other parts of the body, but they usually soon clear up with bland applications.

If the leg is very moist it may be painted with a mixture of talc, zinc oxide, glycerin, and lime water in equal parts, or with silver nitrate solution (1%). Often there is a superimposed infection of the area and it is useful to apply gentian violet solution (1%) as a paint. With dry eczema Lassar's paste spread on thinly with the finger-tip is best. Hydrocortisone ointment or lotion may be helpful but is unnecessary.

Two main reasons for difficulty in the cure of varicose eczema are failure to get rid of the oedema and over-treatment (especially with irritant preparations or such as may provoke allergic sensitization). It must never be forgotten that constitutional causes are likely to be playing a part.

Fusus Oil

Q.—*In shops where diesel fuel injector pumps are tested a special oil called "fusus" oil is often used. When the injectors are being tested the atmosphere in the shop may be quite thick with atomized oil for half an hour, say, at one or two periods during the day. Is this harmful to the workers?*

A.—Shell fusus oil is a highly refined petroleum oil of low aromatic content. The main reason for using it for the type of work in question is that when gas oil (for diesel engines) is used for fuel pump calibration it tends to give rise to deposits if the pump is then placed in storage for a long period.

There have been some instances of dermatitis where gas oil may have been a contributory factor. It is possible that

fusus oil might similarly affect some susceptible individuals. There is little information on occupational exposures to mists of gas oil or fusus oil, but one case of chronic lipid pneumonia arising from the inhalation of mineral oil has been reported.[1] It is considered sound practice not to expose workers to a mist of Shell fusus A, and tests on injectors are best carried out under a hood with forced draught. Barrier creams are generally available for the use of employees exposed to the oil.

REFERENCE

[1] Proudfit, J. P., Van Ordstrand, H. S., and Miller, C. W., *Arch. Industr. Hyg.*, 1950, **1**, 105.

High-fat Slimming Diets

Q.—*Is it true that the high-fat low-calorie diets recently advocated for slimming have advantages over the more usual reducing diets?*

A.—In the present state of our knowledge it is undesirable to use high-fat low-calorie diets for the purpose of reducing weight. The main features of a reducing diet should be to restrict the carbohydrates, protein and fat being allowed in normal amounts. Certain patients who do not lose weight on these diets can be treated with the high-fat regime.[1,2]

REFERENCES

[1] Kekwick, A., and Pawan, G. L. S., *Arch. Middx Hosp.*, 1953, **3**, 139.
[2] —— —— *Lancet*, 1956, **1**, 155.

Gustatory Sweating after Sympathectomy

Q.—*Shortly after bilateral cervical and lumbar sympathectomies eight years ago a woman began to experience sweating while eating. Though slight at first, the sweating has become severe enough to cause much discomfort and social embarrassment. Sweating is most marked after savoury and meat dishes, but occurs to some extent at all meals. It affects the face, scalp, neck, and upper chest, and is at times very profuse. Is this a recognized complication of sympathectomy, and what treatment is advised?*

A.—Gustatory sweating is a well-recognized phenomenon. Like other rarer clinical curiosities such as "crocodile tears" and "gustatory gooseflesh," it has aroused controversy about its physiological basis but no agreement. Three types are known. The first occurs in some normal individuals when they eat certain types of food. The sweating is often accompanied by vasodilatation or lacrimation and is believed by some to be a parasympathetic function like blushing. The second type follows wounds in the neck or face, often in the region of salivary glands, and the third may follow operations on the sympathetic nervous system. An example of the second type was described recently by Young.[1]

In the last type—i.e., following sympathectomy—thermoregulatory sweating is absent. Warming the patient causes no sweating in the denervated area. This means that the sweat glands in the area have lost their sympathetic nerve supply, and they might therefore be expected to become supersensitive to acetylcholine, their usual chemical exciter. If then acetylcholine, liberated by the parasympathetic nerves such as those concerned with blushing, etc., on the face, reached these supersensitive sweat glands, it might activate them. Another possibility is that collateral budding might take place from the parasympathetic nerves to the degenerative sympathetic nerve fibres in the area and thus innervate supersensitive sweat-gland nerve-endings. Treatment must therefore be directed to diminishing parasympathetic activity, and anticholinergic drugs should be tried. Tincture of belladonna has been tried in the past, but the side-effects are often unpleasant if the dose is large enough to control the sweating. There are many new anti-parasympathetic drugs now available, and a trial of these in varying dosage offers an opportunity for alleviation. Should this fail the patient should be referred to a centre where the investigation and surgery of the sympathetic nervous system are being studied.

REFERENCE

[1] Young, A. G., *British Medical Journal*, 1956, **2**, 976.

NOTES AND COMMENTS

Penile Smarting After Coitus.—Dr. D. M. O'CONNOR (Launceston) writes: As no more eminent person has apparently done so, may I venture to add a suggestion to your expert's reply on penile smarting after coitus ("Any Questions?" January 12, p. 118)? A search for a renal calculus might reveal a cause for the symptom.

OUR EXPERT replies: I have never seen a patient with smarting after coitus where it was due to a renal calculus. I have been in touch with several urological surgeons and they are of the same opinion. In my reply I stated that an examination should be carried out before the first morning micturition and bath, which would detect any urinary infection. If Dr. O'Connor has seen a case of renal calculus where there was smarting after coitus, the symptom was in all probability due to urinary infection with a concomitant prostatitis and not to the calculus.

Sulkowitch and Fantus Tests.—Dr. B. J. BOUCHÉ (London, S.W.19) writes: In the answer by your expert on the Sulkowitch and Fantus tests ("Any Questions?" December 29, 1956, p. 1559), there are two points calling for correction in regard to Fantus's test, viz.: (1) The pipette, after measuring 10 drops of urine, is washed twice, once with distilled water and again with some of the pot. chromate solution as used in the test; this is obviously for further safety. (2) The number of drops of silver nitrate solution required to give a brick-red precipitate, less one, is the correct number of grammes of sodium chloride per litre of urine. In other words, the number of drops used before the change of colour (not necessarily the formation of a precipitate) indicates the number of grammes per litre. These two are quite minor points.

Treatment of Acne.—Dr. G. ENGLER (Panama) writes: Referring to the inquiry about recent advances in the treatment of acne ("Any Questions?" December 1, 1956, p. 1317), I would like to mention the remarkable results after the administration of rauwolfia preparations. We have been using "rautensin CUP," a Columbian product, a whole root preparation of *Rauwolfia hirsuta* containing 100 mg. per tablet. However, other rauwolfia products may well be equally effective. The average doses of 600 mg. per day resulted in startling improvement in our cases within a period of two weeks. Besides the reduction of blood pressure by about 20 to 30 mm., several patients have observed a decrease in libido.

Infantile Gratification Habits.—Professor R. S. ILLINGWORTH (Sheffield) writes: May I suggest another answer to your correspondent's query concerning the 20-months-old child who is sucking a napkin ("Any Questions?" January 26, p. 240)? My answer would be (1) it is normal, (2) it will stop as she matures, provided only that you do nothing about it. It will not cause permanent deformity of the teeth, provided that it stops by the age of 5 or 6, as it will if no effort is made to stop it.

Correction.—The sentence in Dr. A. S. Playfair's letter (*Supplement*, February 2, p. 50): "Twice in his letter Dr. Glaisher assumes . . . that his relationship has been destroyed through the machinery of the N.H.S." should have read: "Twice in his letter Dr. Glaisher assumes . . . that this relationship has been destroyed . . ."

Books of "Any Questions?" and Refresher Course Articles.—The following books are available through booksellers or from the Publishing Manager, B.M.A. House. Prices include postage. *Any Questions?*, Volumes 2 and 3 (8s. each); *Refresher Course for General Practitioners*, Volumes 2 and 3 (26s. 6d. each inland, 26s. overseas); *Clinical Pathology in General Practice* (22s. 3d. inland, 21s. 9d. overseas).

All communications with regard to editorial business should be addressed to THE EDITOR, BRITISH MEDICAL JOURNAL, B.M.A. HOUSE, TAVISTOCK SQUARE, LONDON, W.C.1. TELEPHONE: EUSTON 4499. TELEGRAMS: *Aitiology, Westcent, London.* ORIGINAL ARTICLES AND LETTERS forwarded for publication are understood to be offered to the *British Medical Journal* alone unless the contrary be stated.
Authors desiring REPRINTS should communicate with the Publishing Manager, B.M.A. House, Tavistock Square, W.C.1, on receipt of proofs. Authors overseas should indicate on MSS. if reprints are required, as proofs are not sent abroad.
ADVERTISEMENTS should be addressed to the Advertisement Director, B.M.A. House, Tavistock Square, London, W.C.1 (hours 9 a.m. to 5 p.m.). TELEPHONE: EUSTON 4499. TELEGRAMS: *Britmedads, Westcent, London.*
MEMBERS' SUBSCRIPTIONS should be sent to the SECRETARY of the Association. TELEPHONE: EUSTON 4499. TELEGRAMS: *Medisecra, Westcent, London.*
B.M.A. SCOTTISH OFFICE: 7 Drumsheugh Gardens, Edinburgh.

SUPPLEMENT TO THE
BRITISH MEDICAL JOURNAL
LONDON SATURDAY FEBRUARY 16 1957

CONTENTS

Joint Consultants Committee - - - - - - 65
Central Consultants and Specialists Committee - - 66
Interim Report of Mileage Committee - - - 68
Public Health Medical Officers - - - - - - 68
Civil Supplementary Estimates - - - - - - 68
Registrars Group - - - - - - - - - 68
Government's Obligation - - - - - - - 69
Correspondence - - - - - - - - - 69
H.M. Forces - - - - - - - - - 72
Association Notices - - - - - - - - 72

JOINT CONSULTANTS COMMITTEE

A meeting of the Joint Consultants Committee was held at the Royal College of Surgeons on January 23, under the chairmanship of Sir RUSSELL BRAIN.

Medical Membership of Hospital Boards

Among the matters under consideration was a circular issued by the Ministry on December 31 informing hospital boards that the Minister of Health had accepted the recommendation of the Guillebaud Committee that the medical membership of regional hospital boards should not exceed 25% of the total membership save in quite exceptional circumstances, and advising boards to observe the same principle in making appointments to hospital management committees. The Joint Committee had previously urged the Ministry that the membership of a regional board should include at least five consultants actively engaged in the region concerned, and in addition a general practitioner and a medical officer of health, all nominated by the profession. In a letter from the Ministry informing the Committee of the Minister's decision it was stated that the Minister could not accept this proposal, but that he fully shared the Committee's view that the medical members of the regional boards should consist primarily of active members of the consultant staff in the region, together with a general practitioner and a medical officer of health.

The Joint Committee was profoundly disturbed at the Minister's decision, the more so as it appeared inconsistent with his acceptance of the principle that the medical membership should consist primarily of active members of the consultant staff in the region, who are by no means well represented on regional boards at the present time. The Committee thought it inevitable that the small number of consultants representative of those working in the regions must be still further reduced if the overall number of medical members was to be reduced, while still including representatives of the universities and medical schools, and in addition a general practitioner and a medical officer of health.

The Committee felt strongly that any reduction in the medical membership of hospital boards would of necessity affect adversely the valuable part played by the profession in the administration and planning of the service, and that to place upon a smaller number of doctors the responsibility of serving on the boards and their various committees would make the burden intolerable.

The Committee decided, therefore, to ask the Ministry for an early opportunity of discussing the matter further. It decided also to discuss with the Ministry again the question of medical advisory machinery at regional board level, in the light of the recommendations of the Guillebaud Committee, while making it clear that it did not regard such machinery in any way as an alternative to adequate representation on regional boards.

Hospital Medical Staffs

Representatives from the Registrars Group of the British Medical Association attended the meeting to put before the Joint Committee their views regarding the problems associated with hospital medical staffing as they affected the prospects of registrars and senior registrars. They pointed out that the salary scales recommended by the Spens Committee had been formulated on the assumption that consultant status would normally be reached at 32 years of age. At the present time, however, senior registrars in the main specialties did not usually obtain their first consultant appointment until several years later than this, with a consequent loss in their total life earnings of £1,700 for every year promotion was delayed beyond the age of 32. They urged that the introduction of a new career grade below consultant status would not provide a satisfactory solution of the problem, since it would permanently lower the total life earnings of a proportion of new entrants into the hospital service. They recommended that there should be a gradual run-down in the number of senior registrars which would ensure reasonable prospects of promotion after an average training period of four years. Pending such a balance being obtained, and as a purely temporary expedient, they suggested that the tenure and salary scale of time-expired senior registrars should be extended, and newly appointed consultants given seniority on the incremental scale for every year over 32 at the time of appointment.

Appointments in Venereology

A deputation from the Venereologists Group Committee also attended the meeting to discuss with the Committee the difficulties experienced in the staffing of venereal diseases departments in areas where there is insufficient work to make a consultant appointment in the specialty practicable.

Moral Obligation

The Committee agreed to a proposal from the Ministry that the Appointment of Specialists Regulations should be amended to allow a consultant or S.H.M.O. whose sessions are reduced, or whose contract is terminated through redundancy, to be offered an alternative appointment without the necessity to apply for the vacancy in open competition.

Hospital Private Beds

The question of the steadily rising charges for hospital private beds was discussed, and the Committee decided to prepare a case for the introduction of a moderately priced private bed. Reference was also made to the many anomalies in the schedules of operations contained in the Pay Bed Regulations, and instances were quoted of the way in which these schedules operated to the disadvantage of the patient rather than to his advantage. It was agreed to submit a reasoned case to the Ministry for the abolition of the schedules.

Other Matters

Among other matters discussed were the shortage of radiographers and physiotherapists, domiciliary consultations by pathologists, the attendance of chairmen of hospital group medical advisory committees at meetings of hospital management committees, and the payment of fees for the examination of prospective employees at the request of employers.

CENTRAL CONSULTANTS AND SPECIALISTS COMMITTEE

The Central Consultants and Specialists Committee met at B.M.A. House on February 7, Mr. T. HOLMES SELLORS in the chair.

Executive Committee Report

The executive committee reported its action with regard to income-tax relief in respect of the professional expenses of whole-time hospital medical staff. It was decided to ask the Joint Consultants Committee to press the Government to implement the recommendation of the Royal Commission on the Taxation of Profits and Incomes regarding the rules governing the professional expenses of persons in salaried employment.

Consultations with the Staff Side of Committee B regarding the remuneration of S.H.M.O.s in consultant posts were reported. The Management Side had stated it could not entertain generally the suggestion that S.H.M.O.s occupying such posts should be remunerated on the consultant scale, but would be prepared to discuss circumstances relating to individual posts which the Staff Side brought forward. It was suggested by the executive committee that a notice be inserted in the *British Medical Journal* inviting S.H.M.O.s who came within the scope of the review to apply for a form on which they would give the information necessary for their claims to be examined; that a panel be set up by the central committee to examine the applications and submit to Committee B the names of those who appeared to have a prima facie case to be remunerated on the consultant scale; and if Committee B referred the final selection of cases to a subcommittee an S.H.M.O. representative should be co-opted to that subcommittee. It was emphasized that the only people concerned were S.H.M.O.s worthy of consultant grade and carrying out consultants' work.

These recommendations were agreed to.

A report was received of the action of the Council at its last meeting with regard to the integration of medical officers of health with the hospital service (*Supplement*, February 9, p. 56). The executive committee had considered the matter, and reiterated its view that it could not be accepted that a practitioner should be appointed to the consultant staff of a hospital as of right by virtue of his status and office in some other sphere of medicine and without consultation with the hospital staff. Such an appointment would presumably involve duties and responsibilities which might affect the position of other consultants in relation to treatment and beds, and this could not be accepted without consultation with the hospital staff. With regard to membership of the hospital group medical committee, the Ministry had agreed with the Joint Committee that this was a matter for the hospital medical staff to determine, and this principle could not be waived. There should be the closest liaison between the hospitals and the public health service. There were considerable advantages in inviting the medical officer of health to serve on the hospital group medical committee, but the invitation to do so should be made by, or with the approval of, the medical staff concerned.

The Committee agreed with the views of its executive and that they be transmitted to the Council.

It was agreed that the Joint Committee should be asked to make a strong protest to the Minister on his decision to reduce the overall medical membership of regional hospital boards to a maximum of 25% of the total membership, and to press that each board should include not fewer than five consultants actively engaged, at least one general practitioner, and one medical officer of health.

Administration of Drugs by Injection

Dr. Robert Forbes, secretary of the Medical Defence Union, attended the Committee for the discussion of this item, which arose out of a fatality after an injection of penicillin, as a result of which the regional hospital board had issued an instruction that intrathecal injections must not be administered until the quantities had been checked by another doctor, a State-registered nurse, or a student nurse, and a signature obtained that it had been done.

Dr. FORBES said that his society had been asked for advice on this rule, and its counsel instructed it to tell members who asked for guidance that this was a rule which they should not operate, on the ground that it was a serious interference with clinical responsibilities. It related at the moment to intrathecal injections, but in a short time it might relate to many other forms of injection.

The matter was referred back to the executive for further consideration, the view being expressed that regional boards should not be able to tell medical people how to do their work.

Administration of Hospitals

The Committee again considered, after the reference back of its report by the Council, the questions of equal tripartite hospital administration, recommended by the Bradbeer Committee. The Committee had expressed the view that hospital medical staff must have the final authority in the clinical care of patients, but, subject to this, it supported the system of tripartite administration in the general running of a hospital.

The opinion was expressed that tripartite administration had worked successfully in some of the larger hospitals. Did the doctor wish to take charge of the catering department or to engage wardmaids and other staff? The criticism that the Ministry was trying to take away from the doctor the charge of the patient was not justified. There was a considerable body of opinion which did not accept this view and regarded these proposals as the thin end of the wedge.

Members of the Committee said that tripartite administration was not working so well as seemed to be indicated. It was very difficult to get what was required, and the doctor was not asked what he was going to spend—all this was done by administrative staff without adequate consultation with matrons and consultants. Tripartite administration worked where there were first-class men, but everybody wanted to be completely independent, and it would be multipartite before long.

The CHAIRMAN pointed out that all this was considered by the Bradbeer Committee. The Committee agreed to a statement that tripartite administration was successful in some cases but where it was unsuccessful there should be an investigation, and that there should be no lay dominance over the nursing and medical professions.

Young Practitioners

Mr. H. H. LANGSTON, chairman of the Constitution Committee, explained the proposals of his committee with regard to the setting up of a young practitioners' committee. He explained that the need for this committee was strongly

felt; the matter was considered by Council at its last meeting (*Supplement*, February 9, p. 53); and it had been referred to the standing committee for approval. The proposals were sympathetically received, and it was agreed that the Committee should give provisional support.

At the same time there were recommendations before the Committee for reconstitution of the Registrars Group drawn up by its executive committee, and these would be considered at the next meeting of the Group Council. The Committee supported the proposals.

A resolution from a regional committee stating that hospital junior medical staffs should be represented at all levels of the negotiating machinery, particularly in Whitley Committees, was discussed. It was pointed out that members of these committees represented everybody, that there were many groups, and when the affairs of any particular group were being discussed representatives of that group should be invited to attend at the appropriate stage.

Joint Consultants Committee

Dr. ROWLAND HILL, deputy chairman, presented a report on the work of the Joint Consultants Committee and Committee B of the Medical Whitley Council. The question of the statutory registration of medical auxiliaries was again before the Committee, which adhered to its decision that its acceptance of the scheme was conditional upon the co-ordinating council being given effective control over the registration boards and that medical members of the boards should be appointed from names submitted by the appropriate medical organizations and from no other source. It now seemed certain that the scheme would go through and that medical influence would be in the minority even on the co-ordinating council. The auxiliary bodies proposed to call themselves "professions supplementary to medicine." The opinion was expressed at the last meeting of the Committee that if the Minister implemented the scheme the medical profession should dissociate itself from it.

Strong feelings were expressed on the dangers of allowing medical auxiliaries to treat patients without medical supervision of any kind, some members expressing the view that the profession should have nothing to do with the scheme, and that specialties should engage their own auxiliary staff and train them themselves. A resolution was passed unanimously that the Joint Committee be recommended to dissociate itself from the recommendations made by the Ministry of Health in regard to the auxiliary service. It was also agreed that the resolution should be communicated to the various faculties.

With regard to hospital medical staffing, the Registrars Group suggested that as a long-term plan the number of senior registrars should be allowed to run down until there was a better ratio between the senior registrar establishment and the number of expected consultant vacancies. In the meantime the Group endorsed the view that existing time-expired senior registrars should be given security with an extending salary scale.

It was once again stressed that the real answer to the problem of hospital staffing was a review of hospital establishments starting at the periphery and working towards the centre. The most important point was the possibility of well-trained registrars being put out of employment, for it seemed that there was no solution to the problem unless the number of senior registrars was drastically reduced. It was pointed out that there could only be a reduction of senior registrars if their work was done by consultants and the matter came back to a review of establishment. The Ministry's suggestion of the creation of an assistant consultant group was opposed by the Joint Committee and the Central Consultants and Specialists Committee.

Standards of Board-and-Lodging Accommodation

The Committee received a statement from the Registrars Group executive on minimum standards of accommodation for hospital residents. That body also considered that regular inspection of the accommodation should be carried out on a regional basis by inspecting committees appointed by the regional hospital boards. The Registrars Group asked the Committee, if it approved, to forward the proposals to the Board and Lodging Subcommittee of Committee B. The CHAIRMAN said that a subcommittee of the Whitley Committee was discussing what methods were best for establishing throughout the country satisfactory standards of board and lodging. Quite apart from the cost, if the standards were bad there should be a right of appeal against them to the Whitley machinery.

It was agreed that the standards set by the Registrars Group should be put before Committee B.

Future of the Chest Services

The memorandum of the Chest Services Subcommittee, which was set up in January, 1956, to consider and report upon special problems affecting the chest services, was received. It had already been considered by interested committees, which had endorsed the general principles contained in it. The attitude of the Ministry was that tuberculosis was finished and that nothing more need be done about it. Chest physicians did not agree with that view, and put forward proposals for maintaining an organized tuberculosis service based on area clinics. It was agreed that the document should be forwarded to the Joint Consultants Committee.

Remuneration for Part-time Work

The recent agreement for the remuneration of medical practitioners undertaking part-time work for local authorities was reported, and a schedule submitted by the Treasury of fees for work for Government departments not covered by this agreement was considered. The proposals were considered to be satisfactory except in relation to the proposed fee for attendances at pensions appeals tribunals, which should attract a fee of £10 10s. for a day and £5 5s. for half a day's attendance.

The Committee also considered a report from a subcommittee of the Non-Professorial Group Committee relating to the remuneration of university medical teachers and research workers. A memorandum had been prepared by the subcommittee, which had adopted the resolution of the A.R.M. that the remuneration of medical men and women should be that of doctors and should not be related to that of lay personnel. The medical teacher was at a disadvantage because of the later age at which he could commence work, and his salary should be weighted to offset this disadvantage. The figures put forward related to the present time and without prejudice for the near future. The differential between clinical and non-clinical staff was also considered.

The executive committee was authorized to study the memorandum and to transmit it to the Council of the Association for approval so that it could be sent to the university authorities.

The question of fees for examinations carried out in prisons was discussed. It was felt that such examinations could not be classified as domiciliary examinations and that they should carry a minimum fee of £5 5s. for each examination.

Defence Trust

During the day a meeting of the Hospital Medical Staffs Defence Trust was held. The honorary treasurer, Mr. L. DOUGAL CALLANDER, reported that subscriptions amounting to £19,349 were received up to the end of 1956, and interest yields amounted to £411, against a total expenditure of £5,326. The Fund's investments totalled £10,866, and there was on deposit and current accounts £3,568. The last appeal had resulted in a considerable number of cash payments and new bankers' orders, substantially increasing the annual income of the Fund. It was suggested that further appeal letters should be sent out and that action might be taken through the regional committees to stimulate support for the Trust Funds.

Mr. CALLANDER then reported on the action taken by the British Medical Guild in relation to the present remunera-

tion claim. The finances for the Guild had to be found by the defence trusts, and the General Medical Services Committee had already made a very large allocation to the Guild's funds. Not a great deal had been used so far, but if there was an *impasse* after the forthcoming meeting with the Minister there was no knowing what expense would be incurred. He would like to ask for power to transfer £1,000 of the Trust Fund to the Guild funds if required.

After some discussion the proposal was agreed to, subject to consultation with the chairmen of the Joint Committee.

INTERIM REPORT OF MILEAGE COMMITTEE

CHANGES RECOMMENDED

A committee was appointed by the Minister of Health in 1950 to "examine in detail the distribution of the Central Mileage Fund for England and Wales and to make recommendations." The committee, on which were representatives of rural practitioners, has now published an interim report.

The mileage fund is a fund used to recompense rural general practitioners for the loss of earning time incurred in visiting National Health Service patients in country districts over and above that incurred by an urban practitioner for the same number of patients. At present about 87% of the fund goes to meet those expenses that can properly be measured by distances travelled, and about 13% goes to provide additional remuneration where the difficulties of the district are of an exceptional nature.

A scheme for mileage payments was introduced into the National Health Insurance scheme in 1914. It continued as part of the arrangements under the National Health Service in 1948. Until the present committee was set up the scheme had not been comprehensively reviewed since 1924. The committee's report, the preparation of which involved much detailed inquiry, has been sent to all local medical committees and will be part of the G.M.S. Committee's report to the Annual Conference of Local Medical Committees, where it will be open to debate.

Recommendations

The committee anticipates that its recommendations, being of an interim nature, may need amending after experience. In summary, some of the main changes in the present mileage fund scheme proposed are as follows. The fund should be distributed as a whole and called the Rural Practices Fund. Executive councils and local medical committees together should advise the Minister whether they wish the whole or any part of their areas to be included in the mileage scheme. Borough and urban district council areas with populations of more than 10,000 should normally be excluded and rural district areas normally be included. Variations on this should be allowed with the Minister's consent, and a central body should be set up to enable the Minister to consult the B.M.A. and other bodies on whether special requests for variation should be accepted. Patients in designated rural areas should count for payments from the Rural Practices Fund if they live not less than three miles from the doctor's main surgery, except that no doctor should receive any payments unless 10% of his patients live in a designated area and not less than three miles from his surgery.

Units of mileage should be credited on a new scale of one unit for not less than three miles and not more than four miles, and two units per mile thereafter. A model mileage scheme should be prepared by the Minister of Health and the B.M.A., and no variations on this should be allowed without the Minister's approval after consulting the B.M.A. No change is recommended in the present scheme for payments for visiting maternity cases.

The three representatives of executive councils on the committee recorded in the report their "keen disappointment" at the inconclusive results of the committee's deliberations.

PUBLIC HEALTH MEDICAL OFFICERS

INCREASED CAR ALLOWANCES

Committee C of the Medical Whitley Council has agreed on increased rates of motor-car allowances for public health medical officers in England and Wales in view of the circumstances resulting mainly from petrol rationing and the increased price of petrol. The allowances should operate until the end of the month in which petrol rationing stops, and will then be reviewed again. The revised rates take effect from January 1, 1957, and are set out in the appendix to M.D.C. circular No. 33.

The following casual user allowances replace those set out in paragraph 1 of the appendix to M.D.C. circular No. 29: Not exceeding 10 h.p. or 1,199 c.c., 9¼d.; exceeding 10 h.p. or 1,199 c.c. but not exceeding 12 h.p. or 1,399 c.c., 10¾d.; exceeding 12 h.p. or 1,399 c.c. but not exceeding 14 h.p. or 1,599 c.c., 11¼d.; exceeding 14 h.p. or 1,599 c.c., 1s. 0¾d. The essential user allowances, replacing the lump sum allowances and rates per mile in paragraph 2 of the appendix to M.D.C. 29, are as follows:

	Not Exceeding 10 h.p. or 1,199 c.c.	Exceeding 10 h.p. or 1,199 c.c. but Not Exceeding 12 h.p. or 1,399 c.c.	Exceeding 12 h.p. or 1,399 c.c. but Not Exceeding 14 h.p. or 1,599 c.c.	Exceeding 14 h.p. or 1,599 c.c.
Lump sum allowance ...	£66	£72	£78	£84
Rate per mile for first 7,200 miles p.a. ...	5¾d.	6¾d.	7d.	8d.
Excess over 7,200 miles p.a.	4d.	4¾d.	4¾d.	5¾d.

An "essential user" temporarily stopping duty journeys as a result of petrol rationing should get the lump sum allowance in full up to April 30. If rationing continues after that 50% of the lump sum should be paid for the following three months, further payment after that being at the employing authority's discretion.

CIVIL SUPPLEMENTARY ESTIMATES

LARGE HEALTH SERVICE INCREASE

Provision for an extra £26,900,000 net for the National Health Service is made in the Civil Supplementary Estimates for the current financial year. Of this, £15m. is required for hospital running costs, £8m. for drugs, appliances, and medicines (after taking into account an extra £1,200,000 received from patients as a result of the 1s. for each item prescription charge), £2,900,000 for payments to doctors, and £1,600,000 to dentists. Welfare foods, including milk, account for another £1,400,000. An annotation on the Supplementary Estimates appears on page 394 of this week's *Journal*.

REGISTRARS GROUP

PROPOSALS FOR RECONSTITUTION

Thirteen out of the 16 regional registrars groups have approved in principle the Registrars Group Council's proposal that the Registrars Group should be reconstituted to represent all grades of hospital junior medical staff (see *Supplement*, December 8, 1956, p. 209). Three regional groups are opposed to it, and three have not replied to the request from the Group Council for their views. The effect of the Group Council recommendations would be that all hospital staff of the grade of senior registrar and under would be able to take part in regional activities, and that their special interests would be represented at central meetings. The group would be renamed the Hospital Junior Medical Staffs Group. Representatives of the regional registrar groups will discuss the recommendations in the Group Council meeting in May.

GOVERNMENT'S OBLIGATION

Two meetings held on February 8 passed resolutions drawing attention to the Government's obligation to stand by the Spens reports.

The Glossop Division of the B.M.A. resolved that "this Division is firmly of the opinion that the Government has a moral obligation to implement the recommendations of the Spens Committee. The Division further deplores the Minister's evasion of this responsibility. A copy of this resolution to be sent to our Member of Parliament." It was the unanimous feeling of the meeting that all other Divisions in the country should bring similar resolutions to the notice of their respective M.P.s because it was only by such action by individual Divisions that the attention of M.P.s generally could be focused on this urgent matter.

At a very well attended meeting of the British Medical Guild in the Hexham Divisional area the following resolutions were passed: (1) that this meeting is firmly behind the B.M.A. in pressing the Government to discharge its obligations to maintain the standard of medical remuneration in accordance with Spens (passed with one dissentient); (2) that this meeting is prepared to support any action considered necessary even to the extent of withdrawal from the N.H.S. (passed with two dissentients).

Correspondence

Because of heavy pressure on our space, correspondents are asked to keep their letters short.

Unity of Purpose

SIR,—During the past weeks, doctors have held meetings throughout the country to prepare the way for action on behalf of their case against the Government. It is opportune, then, to discuss what measures are available to us. Most doctors would carry out measures such as refusing to sign certificates, pay income tax, and withdrawing from committees, since these would not inconvenience us much and would provide a practical demonstration of our discontent. But these would very likely prove ineffective, as the inconvenience caused could be suffered or lessened by emergency legislation without major disruption of the Service. Failure would leave the profession open to ridicule. The public would suffer considerable irritation and would be likely to be more antagonized than by the adoption of stronger measures. Small measures and small cause for discontent are synonymous to them.

Resignation would be effective if carried out by the majority. We have an excellent case to present to the public in support of resignation. There would be no refusal to treat the sick, or "strike" action. If resignation was associated with appropriate publicity, public opinion would probably rebound against the Government. But unity of action as against promise of action cannot be relied on. There is a fear that those who resigned would be martyrs to a lost cause. To sum up, the profession would be demoralized by the adoption of paltry, undignified measures doomed to failure. On the other hand, the case for resignation is strong, and the mood of the profession shows that the time is opportune.

There has for long been grave discontent among young doctors, who find it financially crippling to specialize and now almost impossible to establish themselves in general practice. Little wonder that the more ambitious—and often the best—emigrate. There is a very real danger of unemployment in the profession under present conditions, and a pool of unemployed or underpaid doctors would leave the rest completely at the mercy of the Government. It is only now that established doctors are beginning to feel the pinch that unrest is widespread and action is considered.

We are not going to get a general 24% pay rise without action. For action to succeed we must have unity. It is doubtful whether unity can be attained on the remuneration aspect alone. There are many doctors who are not prepared to resign over remuneration *per se*, but who cannot see any future for themselves under the present scheme or are constantly irritated by aspects of it. Thus two groups have arisen—those who wish to press the present claim without mention of revision of the Service, and those who wish to revise the Service and hope to improve remuneration and other grievances in so doing. Any course of action must appeal to as much of the profession as possible, for in unity lies our strength. Now that we have travelled so far along the remuneration road let us proceed to the end and the inevitable impasse—and then call for revision of the Service. I do not believe that a new Service would take long to organize. What works in Australia, with evident satisfaction to all concerned, could just as easily work here. If the Government refused to operate a new scheme resignations could be called in; the refusal of the Government to co-operate with us on both remuneration and modification of the Service would strengthen our case. There is much to be said for killing two birds with one stone.

It is most important to study the Government attitude. Assuming that its main interest is in economy and votes, its reaction to a threat of resignation will be to attempt to split the profession. It will, therefore, either postpone matters with an evasively worded comment on Spens and a promise to review the claim when the economic position improves, or offer less than the amount asked for to the whole or part of the profession. Recognition in advance of the motives behind such moves and an alternative scheme, practical and better than the present one, offer the only sure defence against such temptations. Those of us who are content under the N.H.S., and may even have benefited from it, should pause for a moment and consider whether it is not their bounden duty to make medicine an attractive career once more, and not a vocation only.—I am, etc.,

Stockport. C. ALWYN SHARPLES.

SIR,—Two outstanding grievances are noticeable from the correspondence: underpayment and underemployment. The voice of the latter has the handicap of not being effectively heard, and the consequent amount of National Health fodder continues to grow unchecked. Many other causes of dissatisfaction outside these headings exist.

It therefore follows that those who seek redress in one direction alone are unlikely to have the backing of others whose claims are to them equally in need of recognition. If medicine is to survive, and I say this advisedly, for the nature of its present appeal will come to be recognized as ground-bait ultimately and consequently to be shunned, then these serious disaffections need remedying. It is obvious that importuning or legal pressure is not the answer. A system has been created whose members can only be protected by their unity of action. Such unity is impossible to secure unless major causes for dissatisfaction are redressed. It behoves us therefore to recognize each and every reasonable cause of complaint, and not to use the system of committees to silence them. Until we secure a charter of our rights subscribed to by at least 95% of the profession we will continue to be held to ransom. Thereafter, with this unity of purpose defined and agreed, its implementation would turn on a plan drawn up on some such lines. Every member (at least 95%) of the profession would pledge himself without equivocation to support such a charter. He would guarantee such action, practically, by entering into a contractual agreement to back it by as large a subscription as possible—in the nature of a strike fund—if called upon to make it; further, he would agree to forfeit a substantial sum should he default in supporting any action taken to insist on the observance of any clause of the charter.

The suggestions above incorporated have a strict parallel in the trade union movement, so far as I can appreciate it, and are specially drawn to cover (1) the conflicting interests within medicine, and (2) its special need of large funds to meet the circumstances of the doctor's station in life. Dog eating dog and "the orl right Charley" attitude are anachronisms when the profession as a whole is faced with mounting disaster.—I am, etc.,

Worthing. JOHN SOPHIAN.

Medicine and Politics

SIR,—When membership of the Cabinet is changed the papers carry formal letters to and from the Prime Minister. In one of these (*The Times*, January 17) the phrase appeared: "Some changes would be good in order to give experience to those who have not had ministerial appointments." Of course the oncoming politician must be trained and tested, but it is not pleasing to think that the Health Service, we who serve and the patients we treat, may be used as guinea-pigs for this purpose. Is this another argument for the reorganization which will get us all out of politics?—I am, etc.,

Lewes. NOEL H. M. BURKE.

Reform of the Health Service

SIR,—At a crowded gathering of the profession convened to form a local committee of the Guild I was saddened to see how little my colleagues were affected by the *depth* of the crisis facing us. Pay was the great topic. When I warned members of the danger of taking such an unrealistic view of our troubles I was told I was an idealist and that the important thing was to "rake in the lolly." It seems quite tragic that now, when the profession is united and strong (because it is angry), we should waste our powers on the exclusive problem of pay.

I was told that to confuse the reform of the Service with the problem of pay would only cause disunity in the ranks. I cannot see how this can be so. I believe the following basic principles of reform would command the approval of the majority: (1) the removal of the Service from the expedient fingers of the politicians; (2) the immediate payment of compensation at present values; (3) an "item of service" payment; "free" advice has never been thought good advice; (4) return of goodwill—giving back to us a measure of professional freedom; (5) only essential drugs free to all; (6) payment for minor operations and extra payment for being present at a birth; (7) freedom of choice of doctor restored to the patient. Reform is far and away our more important problem. The serpent is writhing on the staff, but an apple will not keep it quiet.

By our calling we are individualists and not given to group loyalties: but who knows when we shall be united again? The future is in our hands, and in this great crisis the eyes of the young in the profession are watching us with hope—and perhaps with fear. This tide in our affairs must be taken at the flood.—I am, etc.,

Lancaster. FRANK S. RICKARDS.

SIR,—I feel that it is a tragic blunder that battle should have been joined with the Minister of Health on the narrow issue of a common wage claim alone, differing in no manner at all from that of our fellow labourers the railwaymen and the dock-workers, etc. What shred of prestige was left the poor general practitioner after Aneurin Bevan had finished with us has now been gratuitously cast away by our ill-chosen and much-publicized approach to the Minister of Health. Not only have we lost status by our methods of approach, but, whereas our brethren of the railroad and dockyards in their wisdom have seen to it that they have previously armed themselves with an iron fist before they have attempted to shake it in anger, we have, in our folly, resorted to precisely the same methods without any power at all to back up our attitude. By the exercise of but a small amount of art, grace, and wisdom, we should most probably have gained all, and certainly more than we are now seeking, and also have exalted our status in the eyes of public opinion, with its manifold advantages, if we had approached the Government at this time with a determined demand for a thoroughgoing and long overdue overhaul of the many defects and injustices of the Service. It could have been made publicly known that it was being done in the interests of both the profession and the public alike, instead of advertising ourselves merely as the hawkers of just another dreary wage claim.

I suggest, however, that Sir Russell Brain has already at this late date opened the door to a wider and wiser approach, and it is my suggestion that the strongest measures possible should be adopted to enforce if necessary, not a wage claim alone, but a demand to the Minister for a reorganized Service, using as a spearhead for our demands changes that will result in a better service to the public and fairer terms with a far wider scope of paid work for the general practitioner than is at present possible. On the other hand, conscientious doctors as a whole do not want the National Health Service Act repealed, they want it drastically overhauled. A mere increase of the present capitation fee would mean that the G.P. with little to offer would then be overpaid again at the expense of his colleague with more to offer. What is required is scope for payment to those practitioners with the training and equipment to increase their service to their patients which would reduce the hospital shambles and facilitate a reduction of the money squandered in this direction. General practitioners do not want their lost status restored by Act of Parliament; the general practitioner will regain honour where honour is due if he is given the opportunity to render work to the best of his ability.—I am, etc.,

Audley, Staffs. JOHN F. JENKINS.

Remuneration Claim

SIR,—May I make an attempt at some constructive criticism in order to help solve some of the difficulties with remuneration? The present system of loading, which makes the 501st patient worth 10s. more than the 499th, could be altered in favour of payment more suited to modern trends. We all know that modern medicine is becoming more a matter of increased work in both geriatrics and paediatrics. Therefore, let us have the scales loaded in favour of those patients who take most of our time—namely, the under twelves and over sixty-fives. As an example (to be worked out in detail by the actuaries), we should be paid 25s. per patient in this group and 20s. for every other patient. Administrative difficulties could be helped by the use of a different-coloured card for this group in their record indexing system. If any further loading is considered possible, any doctors with more than 1,000 coloured cards would be entitled to a loading of 5s. per additional patient. This will encourage doctors to take more elderly patients on their lists.

I hesitate to make any further suggestion with the object of saving the country money, but I do feel that a charge for stay in hospital of £1 per adult and 10s. per child per week is not beyond the average pocket to-day, and there is always the public assistance for those who really cannot afford the charge. Furthermore, a charge for night calls from 10 p.m. to 6 a.m. of up to 10s. 6d. would discourage those who wait as long as possible before sending for the doctor. This charge could be made at the discretion of the doctor—who could waive or lower it according to the circumstances he knows the patient to be in. These ideas are put forward for constructive discussion at future meetings, as I feel they may have some value.—I am, etc.,

Woodford Bridge, Essex. L. M. RUBENS.

SIR,—It is to be hoped that the machinery now being taken out of mothballs includes an effective public information service, lest our preparation for action be met by a nasty popular reaction. Wherefore are we proposing to strike? As I see it, we are proposing to withdraw our services in support of one or other of three alternative demands.

(1) We are claiming a straightforward pay increase of 24%. If this is refused we shall strike—just like any other trade union. If that is our position I, for one, remain a dissentient, since I regard it as an open invitation to other unions to do the same. Unhappily, I believe many lay people regard it as our true position. It is an invidious one, which we must disclaim with all the cogency at our command; for it is inflationary and contrary to the public

interest at this present time. Indeed, we should be setting a better example were we to ask for a pay decrease.

(2) We demand that the Government honour its pledge by implementing the Spens recommendations, wherein our pay was tied to the cost of living. But the Government denies the existence of any such contractual obligation, and, unhappily, legal opinion is divided on the matter. Therefore we cannot say irrefutably that the Government has broken its pledge; we believe it has done so, we may swear passionately that it has done so, but when all is said and done we are in no better position than the plaintiff in a civil action for breach of contract. We may not strike on such an issue, we can only offer to go to arbitration.

(3) We are not claiming a mere pay increase; we are claiming an entirely new method of payment per item of service, a method which is an incentive, not a disincentive as at present. We are pressing this claim in the interests of our patients as well as ourselves. Every day I am embarrassed by patients who apologize for having called me out, who apologize at troubling me for what they regard as minor complaints. That is bad, my friends; it is the reverse of positive medicine. It is distressing to the humble and considerate patient. It is to the disadvantage of the meek.

Dr. C. Glaisher (*Supplement*, January 5, p. 13) has put forward suggestions for a scheme which would, I believe, appeal to the general public, and it is to the general public that we must appeal at this time; for if we win their support we shall not have need of strike action. Public opinion is a mighty force; it is the force that moves governments.—I am, etc.,

Bournemouth. F. ROLAND WILDE.

SIR,—Is it absolutely essential that settlement of our claim should take the form of a proportionate increase all round to offset the reduced value of money? It is all very well to say that redistribution can be considered after our claim has been met, but this line of argument ignores the fact that the lower-paid members of our profession are really up against it and require special consideration *now*. I sincerely hope that negotiators on both sides of the table will keep this fact uppermost in their minds and will see that justice is applied where it is most needed.—I am, etc.,

Belfast. H. J. CRONHELM.

SIR,—Might it not be that the Minister agrees the moral justice of our claim, but cannot, at this juncture, either economically or politically, accede to any large increase in pay from whatever source the demand comes? Such a course would inevitably lead to the devaluation of the pound, with all its subsequent ills. That the Service is unsatisfactory and in part degrading cannot be denied, but I cannot help thinking that the removal of the financial barrier as between the sick and their treatment is one of the greatest blessings that this country has ever received. Many would have us resign from this *en masse*. Where would we go from there? Would we go back to bills, and the cutting of treatment down to the minimum so as to avoid hardship to the patient? One correspondent writes that we are the only nation which has been crass enough to institute such a health service. Might we not be the only nation (except Russia) which has had the courage to do so?

Some advocate payment per item of service. Would this not be again erecting the financial barrier? From where is the extra money to come so that the doctors may be better off? Presumably, in part, out of their own pockets in the form of extra taxation. Have we not enough taxation already? This would force more people to apply for public assistance. Already one person in every fifty in this country is supported by public assistance. I must disagree with Dr. W. R. Blatchley (*Supplement*, January 12, p. 20) when he writes: "What doctor worthy of the name could not make a better living . . . in private practice?" My answer to him is that thousands of doctors are to-day financially better off than they were pre-N.H.S.

I believe that eventually we shall find that the only real solution is a salaried service, provided that the public can be sufficiently safeguarded. We should then be in a position where we could afford to be united in pressing our claims: we should not be imprisoned in one practice for the whole of our professional life: we should not have to provide our own surgeries: we should work an eight-hour day: our pay would be secure and not at the mercy of a more proficient competitor.—I am, etc.,

Cheriton Bishop, Devon. F. E. GRAHAM-BONNALIE.

SIR,—I suggest we stop these threats of strikes to secure our just demands. Instead let us embarrass the Government by economies. The first of these should be our telephones. On a fixed date we should inform the Postmaster-General we wish to have our telephones removed. The confusion within the Service would be considerable, yet at no loss to us. This last factor will be appreciated by those, like myself, who cannot afford to strike.—I am, etc.,

Irvine. ANDREW A. GARVEN.

P.A.Y.E. and Surtax

SIR,—Not so very long ago many members of the consultant staffs of the hospitals of this country were transferred, willy-nilly, to the P.A.Y.E. system of tax collection. The system has some virtues, but as applied to this body of men and women it contains an injustice. With surtax at its present level a large proportion of these people are liable to this specific tax, which is levelled upon the total taxable income above £2,000 per annum. Now a man subject to P.A.Y.E. never handles his total earnings—he cannot invest it or put it to work for him in any way. In fact part of his income is a polite fiction. Surely he should only pay surtax on that part of his income over and above £2,000 which he does actually receive. The coexistence of P.A.Y.E. and surtax, as applied to the same individual, cannot be defended in equity or ethics.—I am, etc.,

Birmingham. L. T. CLARKE.

Medical Trade Union

SIR,—One of the weaknesses of our present negotiating position is that we have no access to arbitration except as a concession from the Minister of Health. Our relative success with the Danckwerts award makes it unlikely that the Minister will agree to arbitration again.

At a recent meeting of doctors in Manchester called to reorganize the Medical Guild a speaker pointed out that if the Guild were a trade union then it could have our present dispute referred to an industrial disputes tribunal, and in this way could secure arbitration. However, we learned from the platform that the Guild is not a trade union in a legal sense and cannot take this action. We all know that by its Articles of Association the B.M.A. is precluded from acting as a trade union, but I do not know of anything in those articles which would preclude it from sponsoring a trade union. It would be a relatively simple procedure to circularize members with a form of application to join the British Medical Union, if we decided to call it that, and it could be organized in a very short time using the B.M.A. offices and staff. I visualize such a union as being a legal device to secure arbitration if negotiations break down, and I do not suggest that any of the more undesirable of trade union activities should be contemplated.

At present we are trying to fight a battle without weapons, and I have no confidence in threats of mass resignation and so on, nor do I agree with the confusion of reforms in the N.H.S. with the question of remuneration. We should have one objective at a time and should make certain we gain the first objective before going on to the second. The proposition I have outlined above seems to me well worth considering, and I would welcome comments on it from a B.M.A. representative and, even more, from a legal expert.—I am, etc.,

Manchester 8. C. J. GRIFFIN.

H.M. Forces

A *Supplement* to the *London Gazette* has announced the following appointment and mention in dispatches in recognition of distinguished services in Cyprus for the period July 1 to December 31, 1956:

O.B.E. (Military Division).—Lieutenant-Colonel K. F. Stephens, R.A.M.C.

Mentioned in Dispatches.—Captain I. R. D. Reid, R.A.M.C.

ROYAL NAVY

Surgeon Lieutenant-Commander C. E. Wells has terminated his commission.

ROYAL NAVAL VOLUNTEER RESERVE

Surgeon Lieutenants M. Wickham, M. G. D. Davys, A. F. Murphy, W. A. Copland, P. W. Harvey, R. L. H. Barnard, C. V. Kaula, M. W. Eddings, and W. J. C. Scott to be Surgeon Lieutenant-Commanders

ROYAL ARMY MEDICAL CORPS

Captains J. P. Crowdy and A. P. B. Harston to be Majors.

REGULAR ARMY RESERVE OF OFFICERS

Major-General J. M. Macfie, C.B., C.B.E., M.C., late R.A.M.C., having attained the age limit of liability to recall, has ceased to belong to the Reserve of Officers.

ROYAL ARMY MEDICAL CORPS

Captain (Acting Major) I. L. Gregory, from A.E.R.O., to be Captain, and has been granted the honorary rank of Major.

Class III.—Captain (War Substantive Major) (Honorary Lieutenant-Colonel) W. G. MacDougall, having attained the age limit of liability to recall, has ceased to belong to the Reserve of Officers, retaining the honorary rank of Lieutenant-Colonel.

BRITISH MEDICAL GUILD

Meetings to be Held

MARYLEBONE.—At Committee Room C, B.M.A. House, Tavistock Square, W.C., Friday, February 22, 8.45 p.m.

TOWER HAMLETS.—At Recreation Hall, Mile End Hospital, Bancroft Road, E., Thursday, February 21, 8.30 p.m.

Association Notices

Diary of Central Meetings

FEBRUARY

20	Wed.	Representatives of Negotiating Committee to meet new Minister of Health (at 23, Savile Row, W.), 11 a.m.
20	Wed.	Catering Committee, 12 noon.
20	Wed.	Arrangements Committee (Birmingham, 1958), 2 p.m.
20	Wed.	Negotiating Committee, 2.30 p.m.
21	Thurs.	G.M.S. Committee, 10.30 a.m.
22	Fri.	Staff Side, Committee C, Medical Whitley Council, 11 a.m.
22	Fri.	Public Health Committee, 2 p.m.
22	Fri.	Subcommittee on Medical Reports, Joint Committee of B.M.A. and Magistrates' Association, 2.30 p.m.
28	Thurs.	Compensation and Superannuation Committee, 2 p.m.
28	Thurs.	Non-professorial Group Committee, 2 p.m.

MARCH

1	Fri.	Assistants and Young Practitioners Subcommittee, G.M.S. Committee, 2 p.m.
4	Mon	Alternative Edition Subcommittee, Joint Formulary Committee, 11 a.m.
5	Tues.	Editorial Subcommittee, Joint Formulary Committee, 11 a.m.
7	Thurs	Charities Committee, 2.30 p.m.
8	Fri.	Overseas Committee, 2 p.m.
21	Thurs.	G.M.S. Committee, 10.30 a.m.

Branch and Division Meetings to be Held

ABERYSTWYTH DIVISION.—At General Hospital, Aberystwyth, Sunday, February 24, 3 p.m., business meeting.

BIRMINGHAM DIVISION.—At 154, Great Charles Street, Birmingham, Tuesday, February 19, 8.30 p.m., meeting. Lecture by Professor W. Melville Arnott: "Drugs—Their Uses and Abuses."

BOURNEMOUTH DIVISION.—At Board Room, Royal Victoria Hospital, Boscombe, Friday, February 22, 8.15 p.m., meeting. Address by Mr. P. W. Clarkson: "Contribution of Plastic Surgery to the Treatment of Endocrine Disorders."

BRADFORD DIVISION.—At Medical Societies' Room, Bradford Royal Infirmary, Tuesday, February 19, 8.15 p.m., meeting in conjunction with Bradford Medico-Chirurgical Society. Symposium on cortisone.

COVENTRY DIVISION.—At Grosvenor Room, Hotel Leofric, Tuesday, February 19, 7.30 for 8 p.m., combined supper meeting with members of local dental profession. Address by Dr. R. W. H. Tavenner: "Osteomyelitis and the Acute Alveolar Abscess."

DARLINGTON DIVISION.—At Darlington Memorial Hospital, Tuesday, February 19, 8.30 p.m., meeting. B.M.A. Lecture by Mr. R. B. Zachary: "Surgery in Infancy."

EAST HERTS DIVISION.—At Mayflower Hotel, Hertford, Tuesday, February 19, 8 p.m., meeting of Hertford and District Branch of Pharmaceutical Society to which members of the Division are invited. Talk by Dr. J. J. F. Merry: "Therapy with Cortisone" A colour film will be shown.

FINCHLEY DIVISION.—At Board Room, Finchley Memorial Hospital, Granville Road, N. Finchley, N., Friday, February 22, 8.30 p.m., meeting. B.M.A. Lecture by Dr. H. Neville Stafford: "Some Unusual Cases in a Coroner's Experience."

GUILDFORD DIVISION.—At Board Room, Royal Surrey County Hospital, Thursday, February 21, 8.30 p.m., meeting. Lecture by Mr. N. C. Lake: "Problems of Foot Disorders."

HUDDERSFIELD DIVISION.—At George Hotel, Huddersfield, Thursday, February 21, 8 for 8.30 p.m., inaugural dinner.

LONDONDERRY DIVISION.—At City Hotel, Londonderry, Saturday, February 23, 8 for 8.30 p.m., dinner.

MID-ESSEX DIVISION.—At Out-patient Hall, Chelmsford and Essex Hospital, London Road, Chelmsford, Sunday, February 17, 10.45 a.m., meeting. Discussion: "The Menopause." Opening speaker, Mr. A. C. Pearson.

MIDLAND BRANCH.—At Selly Oak Hospital, Birmingham, Friday, February 22, 8.15 p.m., meeting of clinical and pathological section.

MONMOUTHSHIRE DIVISION.—At 20, Stow Hill, Newport, Thursday, February 21, 8.30 p.m., meeting. Address by Professor A. S. Duncan: "Importance of the Placenta in Obstetric Practice." Guests are invited.

NORTH MIDDLESEX DIVISION.—At Committee Room, North Middlesex Hospital, Silver Street, Edmonton, N., Tuesday, February 19, 8.30 for 8.45 p.m., meeting. Paper by Dr. G. I. M. Swyer: "Use of Hormones in General Practice." Medical guests are invited.

NORTH STAFFS DIVISION.—At Grand Hotel, Hanley, Tuesday, February 19, 8 p.m., supper meeting. Lecture by Dr. E. M. Donaldson: "The Dermatitis Myth."

OLDHAM DIVISION.—At Albion Club, Queen Street, Oldham, Monday, February 18, 9 p.m., meeting. Dr. C. S. Don: "Treatment of Diabetes—Some Recent Advances."

ST. PANCRAS DIVISION.—At Committee Room C, B.M.A. House, Tavistock Square, London, W.C., Wednesday, February 20, 8.30 p.m., meeting. Lecture-film-demonstration by Dr. P. Sattin: "General Practice Organization." Hospital officers, general practitioners, and members of the City Division are invited.

SCARBOROUGH DIVISION.—At Board Room, Scarborough Hospital, Thursday, February 21, 8.30 p.m., meeting. Lecture by Dr. R. E. Bonham-Carter: "Acute Cardiac Illness in Children."

SOUTH SHIELDS DIVISION.—At Ingham Infirmary, South Shields, Thursday, February 21, 8.30 p.m., meeting. Address by Dr. E. Grey-Turner (Assistant Secretary, B.M.A.): "Present Medico-Political Situation." All medical practitioners in the area of the Division are invited.

SOUTHAMPTON DIVISION.—At Conference Room, Civic Centre, Southampton, Wednesday, February 20, 8.30 p.m., general meeting. Address by Dr. Keith Simpson: "What to do with a Dead Body." Members are invited to meet the speaker at dinner in the Polygon Hotel at 7 p.m.

SOUTH-WEST ESSEX DIVISION.—At Langthorne Hospital, Langthorne Road, E., Wednesday, February 20, 8.30 p.m., general meeting. Discussion to be opened by the Bishop of Barking and Dr. Eric Ashton: "Divine Healing and Co-operation Between Doctors and Clergy." Clergy resident in the area of the Division are invited.

SWANSEA DIVISION.—At Osborne Hotel, Langland, Thursday, February 21, 7.30 for 8 p.m., dinner. Address by Dr. G. D. Kersley: "Trends in Treatment of Rheumatic Diseases."

TOWER HAMLETS DIVISION.—At St. Andrew's Hospital, Devons Road, Bow, E., Friday, February 22, 3 p.m., meeting. Mr. A. J. Walton: "Surgery of the Neck."

WEST MIDDLESEX DIVISION.—At Feathers Hotel, Ealing Broadway, W., Tuesday, February 19, 8 p.m., joint meeting with West Middlesex Chemists' Association and Branch of the Pharmaceutical Society. Lecture by Dr. F. E. Camps: "Some Problems in Toxicology"

WEST SUFFOLK DIVISION.—At Ball Room, Everard's Hotel, Bury St. Edmunds, Tuesday, February 19, 8.30 p.m., annual general meeting. Talk by Dr. Walter Hedgcock (Assistant Secretary, B.M.A.): "Current Affairs."

Dangerous Drugs Act : Withdrawal of Authorities

The Home Office announces that Dr. John Galbraith Mackay Anderson (Birmingham) and Dr. Esmond Millington (Hove) are no longer authorized to be in possession of or to prescribe those drugs to which the Dangerous Drugs Regulations apply.